MANAGEMENT OF THE SICK INFANT

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PIONEERS OF PEDIATRICS IN CALIFORNIA

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PREFACE TO SECOND EDITION

The reception given to the first edition of this book has been a matter of gratification to the writers, and they wish to express their thanks to the members of the profession who have made the second edition necessary and to the reviewers who have given the work such favorable comment.

In the revision, the attempt has been made to remedy some of the errors and omissions and to incorporate the more important and practical of the things which have developed since the first printing was made.

L. P.
W. E. C.

FOREWORD

The writers have been impressed with the fact that they have been unable to find any text book in the English language which deals exclusively with the peculiarities of disease as it occurs in infants. There appears to be a need for such a work.

It has been our endeavor to codify the things that have seemed to aid us in dealing with sick babies and to present them in this volume; and it is our hope that our presentation may help the practitioner of medicine and through him, be of some service to sick infants.

L. P.
W. E. C.

San Francisco, California.

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MANAGEMENT OF THE SICK INFANT

PART I

CHAPTER I

GENERAL CONSIDERATIONS

The human infant presents many peculiarities of function and structure; these are such that, when sickness overtakes a baby, the management of its case demands many departures from ordinary methods of treatment—methods quite appropriate when the physician has to deal with older patients.

The relative immobility and the helplessness of the infant have to be considered. Thought must be given to the fact that the digestion and metabolism of the child are delicately unstable; that the adjustments of water and mineral salts in the tissues of the very young may be disturbed by very slight influences and that as yet little is clearly known, either about the processes themselves or about the agencies which interfere with them.

The inefficiency of the infant's thermal regulating apparatus provides another physiologic reason why sick infants need special methods of management and care. A still more important reason is the undeveloped state of the nervous system which often is unstable, poorly controlled, oversensitive to external impressions, and subject to too rapid neuronic discharges. This state of affairs leads to the ready development of convulsions and must influence and modify the methods of treatment.

Then too, the methods of treating the very young are necessarily altered by the fact that babies are especially vulnerable to, and must be protected from, the common infections, especially those which attack the respiratory tract. This vulnerability makes a careful control of the child's associates a most important measure of sickness prevention at this age. The possibility of hand-borne infection should always be remembered. No stranger should be allowed to kiss and fondle the baby's hands, for they

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In many of the following chapters of Part I, it may be noted that the predominant symptom alone has been dealt with. For a fuller discussion of a given disease, the reader is referred to the appropriate chapter in Part II.

are rarely long out of the child's mouth and so they become the most ready vehicles for the transmission of contagion.

If it be true that good nursing is the basis of success in treating the adult patient, it is doubly true in the case of the sick infant. At this early age, the physical discomforts of illness are accentuated by the patient's inability to change his position or to call attention to excessive covering, tight clothing or inappropriate bedding. Neither can the little one, not having yet acquired control of his evacuations, indicate the presence of irritating discharges. He may often suffer from thirst because he is unable to demand water. These facts are so obvious that it may seem unnecessary to dwell upon them, but experience teaches that on minute attention to such details is based the difference between good and indifferent treatment.

Many babies, sick and well, are allowed to suffer because of the mistaken idea that soft mattresses and downy pillows are sources of comfort; and yet it cannot escape the attention of physicians that many crying and uncomfortable babies will often cease to fret when they are taken from such a bed and are put upon a hard table. It is plain that soft bedding does not give the proper support and that it may produce cramped muscles as the result of uncomfortable positions which the child is unable to change. Soft bedding also acts as an insulator, retaining heat that should be radiated, and so contributes to make the infant wretched. Such error in technic may have a very grave influence on the fevers of infancy, especially on those associated with respiratory tract infections. In this connection, the superstition that an excess of clothing is essential to the well-being of babies with colds, seems immovably fixed in the minds of the laity. Many a sick child is insulated with pneumonia jackets, extra blankets and comforters and heavy underwear and is denied the advantages of tepid baths, packs and other valuable antipyretic measures.

Even if the bedding is not excessive, it may be so tightly applied that the child is held within it as in a splint; the normal movements of its legs and feet are interfered with, and pressure is made upon the toes, often to its great discomfort. This fault can be avoided in certain cases by supporting the bed covers on a small wire or wooden arch, similar to the ordinary bed cradles that are used for the protection of broken limbs. It should be mentioned here that the all too common practice of having a sick infant sleep with its mother because of the fanciful conception that

the child draws strength from the vigor of the healthy parent, must be sternly repressed.

Although stress must be laid upon the disadvantages of overclothing, it must not be forgotten, however, when we are dealing with premature or emaciated infants, especially with those who have a subnormal temperature, that the application of external heat is a life-saving measure. Such infants have an impaired heat regulating apparatus and need the insulation which might prove fatal to children with high fever. This protection is best afforded by the use of a garment made in the form of a cape and cloak from sheet cotton quilted between layers of cheese cloth.

It is unfortunate that there has been so much controversy in regard to the principles of infant feeding, for the uncertainty that has flowed from these discussions has impaired the confidence of many practitioners in their ability to solve nutritional problems. As a matter of fact, the practical physician should rarely fail to arrive at a correct solution of these difficulties. For the most part, the answer will be found in the knowledge of how to use or to withdraw the various foods that are available; of the state of the child's water balance; and consideration of the character of the intestinal flora. Detailed discussion of these points is undertaken in the chapter on Nutrition.

Consideration of the principles of nutrition is of highest importance, not only when we are studying those children whose illness is referable to disturbances of the digestive tract, but also in any other sickness that may afflict a child during the first year of life. The entire dependence of the child on its mother's breast milk or on some artificial substitute, taken together with the delicate adjustment of the infant's processes of digestion and assimilation, may lead to a maze of difficulties, once illness invades the nursery; for the sick baby may react with pathologic responses even to healthy breast milk—the most appropriate of foods for well babies.

No wiser biological generalization has ever been made nor any more helpful to the understanding of physiological processes than that which considers each individual cell of the animal body a modified marine organism. It follows then, that as such an organism, each cell has special needs, both for fluids and for mineral salts; these needs are supplied by the tissue juices, lymph and blood, which surround the cellular units of the healthy body. The mature cell is supplied with resistances and regulating powers

which give it a wide range of adaptability to changes in the circulating fluids; but the younger the body, the less delicate and effective are these mechanisms. The result is that in the immature, water loss, salt concentrations or impoverishments take place readily and may lead to such profound alterations in metabolism that death must ensue unless the balances are promptly restored. The clinical pictures essentially dependent upon changes of this sort appear most characteristically in the diarrheas. As a matter of fact, when death follows excessive evacuations of the bowels, very often it is directly due to metabolic disturbances, the result of dehydration. Disturbance of water balance also plays an important part in the clinical condition known as "marasmus" and "athrepsia."

Therapeutic measures designed to maintain the water balance of infants have not received the recognition their importance deserves. It is to the merit of Blackfan that he called the attention of the profession in this country to the safety of the intraperitoneal injection of normal salt solution as a rapid and certain method of giving water. If a short-bevel needle is used and reasonable care taken, it is almost impossible to do any damage to the intestine, a fact attested by many observers. Certainly there is no more dramatically effective therapeutic measure available. Often, after an injection has been made into the peritoneum, the recovery of many of the dried-out, apparently moribund victims of diarrhea, vomiting, or alimentary intoxications is almost miraculous.

In severe cases, it may be wise to supplement intraperitoneal injection by intravenous injection of hypertonic colloidal solution. Either 7 per cent acacia or 5 per cent glucose in a normal saline solution is useful when given in this way. Some writers have recommended the intravenous injection of glucose solution in 15 per cent to 20 per cent concentrations, a method which is often brilliantly successful, although these high concentrations have little real advantage over injections of 8 per cent to 10 per cent strength.

Acidosis, often the fatal factor in alimentary intoxication, is accompanied by a greatly diminished renal excretion of acid sodium phosphate and by an accumulation of this salt in the circulation—conditions which raise the hydrogen-ion concentration of the blood, often to a fatal degree. If an adequate subcutaneous or intraperitoneal injection is given promptly to an intoxicated

child, it will dilute the blood, reestablish the excretory power of the kidney, and rescue the patient.

The normal functioning of the body is dependent not only on a normal fluid balance, but also on an appropriate concentration of mineral elements. There is an exquisite mechanism for regulating bicarbonate balance of the tissues through which a very definite relationship is maintained by the CO_2 of the alveolar air and the bicarbonate content of the blood. The minutiæ of the processes which control the concentration of the other minerals in the body, are not so well understood, although we know that the kidneys play a predominant rôle in this matter, while the intestinal mucosa is also an essential factor, especially in the control of calcium balance.

The frequent appearance of edema in infant patients makes it imperative that the pediatrist give due consideration to the intake and excretion of salts, especially of sodium chloride; for there is no doubt that it is upon disturbances of salt balance that excessive water retention depends. Extreme anasarca may follow exclusive whey feeding; and Chapin, Still and other writers have dwelt upon the edemas which follow when barley water and other highly-mineralized cereal decoctions are fed to the exclusion of protein-containing foods.

The subject of water metabolism is very closely connected with the matter of heat control. The loss of water by the lungs and skin is the chief means by which temperature is reduced. Subnormal temperatures often accompany certain of those types of alimentary intoxications in which forced ventilation of the lungs is extreme. The clammy skin and the blue, pinched, slightly cyanotic face of the infant suffering from food intoxication are familiar. Such a baby is often found covered with cold perspiration, an index of changed water metabolism reacting to produce a subnormal temperature. The subnormal temperature of the athreptic child is another instance which illustrates how closely thermal regulation and water balance are related. Some modern physiologists are coming to believe that fever itself is an expression of the body's inability to maintain the balance between water intake and excretion. More and more the belief in an occult, intelligent, thermo-regulatory brain center is falling into abeyance.

The sheltered environment of the infant and its natural immunity protect it to a large degree from infectious diseases but do not protect it from digestive and nutritional disorders nor

from those respiratory infections which are derived from close contact with adults. The result is that such communicable diseases as diphtheria and scarlet fever are rarities at this age; and in those families in which the common cold is recognized as of infectious origin, and proper precautions taken, babies seldom suffer from coryza, bronchitis or pneumonia.

There are certain skin disorders which seem to be essentially of this age—the so-called infantile eczemas, intertrigo and in late infancy, the urticarias of all forms. An influential school of pediatrists regard the infantile eczemas and urticarias as external expression of a general disorder that has been called the "exudative diathesis" which, if it can be accepted as a pathologic entity, may be related to disturbances of water and salt metabolism.

Rickets and scurvy, essentially of this age are also nutritional in origin. If the earlier stages of the former disease were always recognized, undoubtedly there would be fewer cases of bone and cartilage defect in late infancy. It is the writers' belief that the so-called "balance disturbance" baby—the child with sweating head, ill-nourished hair, extreme pallor, dark circles under the eyes, who is at a standstill in weight or shows irregular gain, and who is restless and somewhat tender, is essentially rachitic. At least he is in the early stages of that disorder. It is undoubtedly true that if such children are given a diet and regimen which correct these early toxic symptoms, the skeletal and muscular defects may be forestalled.

One of the most important duties of the practitioner is to recognize the earliest signs of nutritional disturbance and to meet them with a plan of feeding and regimen which will prevent the full development of disease. The prescription of orange juice at the proper time is important, because it will make it unnecessary at a later date to treat a full-fledged scurvy. Careful attention to an anorexia, or a pallor, or a mild head-sweating, and treatment by the withdrawal of excessive amount of milk and the institution of a better balanced dietary, may save a child from bow legs or a woman from a flat pelvis. Intelligent methods of prevention applied during the age of infancy will bear fruit in every subsequent year of the individual's life.

CHAPTER II

VOMITING

Vomiting, so frequent in infancy, must always be regarded as a symptom. It may indicate anything from slight overfilling of the stomach to the gravest conditions based on bowel obstruction, central nervous disease or irreparable toxemia. For systematic consideration, it is well to divide the types of vomiting into classes, and no division seems more useful than that which groups the different varieties as first, *irritative;* second, *toxic;* third, *obstructive;* and lastly *central.*

In the newborn, **irritative** vomiting is a common event. The irritation may be engendered by swallowing maternal discharges. Under these circumstances, the onset is within the first twelve hours and may be slight or severe. The vomitus at first is brownish and consists of altered, partially digested mucous and swallowed material. Later, bile is also ejected, and if the vomiting continues sufficiently long the ejecta become copious and watery, and the child shows evidence of dehydration. To meet this condition, no measure is more effective than stomach lavage. Normal saline solution or 2 per cent sodium bicarbonate solution may be used in the manner described in the chapter on Methods.

Another fertile source of irritative vomiting is the attempt to force too early feeding. This can be prevented if one gives no milk mixtures to a baby during the first 48 hours. A 5 per cent sugar solution at 4 or 6 hour intervals will meet the needs of the child during the first two days of its life, and for even a longer period if the secretion of the breasts be delayed. However, if the physician should have to deal with a case in which the feeding has been obviously improper at this age, a stomach lavage followed by a few feedings of 5 per cent lactose or maltose, made alkaline with a ½ of 1 per cent solution of sodium bicarbonate will usually effect a cure. The feeding should be given only at 4 hour intervals. In the case of premature babies exception must often be made for many of them will vomit the large amounts required to meet their daily water needs if they are fed at long intervals. Such babies should be fed smaller amounts at shorter intervals.

Breast milk or artificial food deficient either in quantity or quality, may be productive of vomiting. In such cases the recti-

fication of the feeding will remedy the emesis. The accuracy of the diagnosis of insufficiency as indicated by loss of weight is sustained when the characteristic scanty, greenish-brown hunger stool is passed.

Emesis due to **toxemia** is a graver matter and frequently results in fatality. Among the least promising of the cases of this group are those arising among the offspring of a toxemic mother. Fortunately not every such mother affects her baby in this way. These infants begin to vomit rather early, in the first twenty-four hours, and the intensity and frequency of the attacks increase from day to day. Nursing at the breast merely aggravates the vomiting, for the ingestion of the mother's milk seems often to render the child more toxic. It is on record that children have died in convulsion after taking one or two nursings from the breasts of toxemic mothers. Stomach lavage alone, while a valuable procedure, will not effect a cure. Limited starvation together with the intraperitoneal injection of normal saline solution, and in the severer cases, injection into the longitudinal sinus of hypertonic glucose solution will be required. When shock is in evidence, especially if the depression is great, 2 to 5 minims of a 1 to 1000 adrenalin solution may be added to the injections. The required dose of the drug will vary with the weight of the child. External heat applied as a simple hot pack, a mustard pack, or in the form of hot water bottles will be needed.

Intractable vomiting may be one of the important symptoms indicating *sepsis of the newborn.* The accompanying fever, or in the very weak infant, a subnormal temperature, together with the discovery of a source of infection, will suggest a course of treatment aimed at eradicating the focus and sustaining the strength of the child. *Streptococcus infections* in the region of the umbilicus running into *peritonitis, infective cholangitis or general infection* by way of the blood stream, are the most frequently encountered. The widespread *infections of the skin* often part of a general infection which terminates in vast desquamations, are almost always accompanied by persistent vomiting. In such cases, the treatment is supporting. Every attempt should be made to raise the resistance of the child by the use of rational formulas. In such cases, much may be gained by using the open cage in a warm bed as described in the chapter on Methods.

Peritonitis in the newborn is a condition diagnosed with the greatest difficulty. If a diagnosis be made and it is certain that the process is localized, then there should be immediate resort to

surgical intervention. The prognosis is of the gravest, as it is also in cases of infective cholangitis and in generalized infections.

Obstructions of any portion of the gastrointestinal tract may result in vomiting. The higher up the interference, the more prompt and insistent will be the emesis. In addition to the ejection of the ingesta, the most striking symptoms of this condition are visible peristalsis and distention of that portion of the tract lying above the obstruction. In duodenal narrowing, a visible stomach wave is identical with that seen in infants suffering from hypertrophic pyloric stenosis. When the stricture is in the jejunum, a limited amount of distention with peristalsis occurs about the umbilicus. Narrowing in the terminal ileum and cecal region is evidenced by a greater distention and a wider spread peristalsis in the same area; while in obstructions about the sigmoid and rectum, not only the large intestine, but also the small, may be seen in active movement.

The most serious lesions which cause obstruction occur in the congenital *atresias* and *stenoses*. Such lesions in the esophagus are reported in medical literature and they are almost unfailingly fatal. However, a radiographic examination is always called for as it affords the only means for determining the operability of the case. The cardiac end of the stomach may be involved in an obstructive malformation, and a similar defect is sometimes found in the duodenum of children who begin to vomit immediately after birth. Obstructions at the latter site are remediable through gastroenterostomy. The records of the Children's Hospital of San Francisco contain a description of a child with a complete developmental failure of the upper portion of the duodenum. A gastroenterostomy was performed on the fourth day of its life. This was followed by an uninterrupted recovery, and the child has grown and developed in a normal way. Not always, however, can one expect so favorable an outcome, because atresias and stenoses of the duodenum may be but part of a condition of multiple stenoses and atresias affecting all the anatomical divisions of the gut.

Microcolon and other inborn anatomic faults of the large intestine may be part of the clinical picture of multiple stenoses, or they may occur alone. In the first instance, vomiting will be late in its onset and fecal in character when it does occur. These symptoms would happen also if similar obstructions limited to the lower end of the large bowel (rectum and anus) had been over-

looked. Under any circumstances, recourse to surgery would be fully justified, although the prognosis is most unfavorable. Should the obstruction be single, or should it involve merely the rectum or anus, surgical procedures offer some hope that the child's life may be saved. But if, as so often happens, the intestine is obstructed at several points, surgery is powerless to aid the infant.

If the obstruction at the lower end of the canal occurs because of a failure of the membrane at the junction of the proctodeum and the hindgut to absorb, relief can be had by simple incision. If, however, there is extensive atresia without proper development of the rectum, a more complicated operation is required. A determination of the type of obstruction must depend on manual palpation. With a simple obstruction, the sensation encountered is that of a flexible, thin and yielding membrane; while with an atresia, the palpating finger meets with an unyielding and resistant mass.

Sometimes, in the presence of vigorous vomiting accompanied by a visible peristalsis, it is difficult to decide whether we are dealing with a true developmental blocking of the gut or with an intestinal obstruction due to an *inspissated mass of meconium*. In either instance, stomach lavage is indicated; and this measure, together with large alkaline enemas, will relieve the obstruction, if the cause be accumulated intestinal contents. One must be careful, however, not to use purgatives unless the diagnosis is beyond all doubt, for vigorous peristalsis will vastly aggravate the condition when it arises from anatomical defect.

Hernias, when obstructed, are an infrequent but possible source of vomiting in the newborn. Diaphragmatic hernias may be responsible for the symptoms which will then be accompanied by much respiratory distress on account of the encroachment on the lungs of the gas-containing abdominal viscera. Surgical intervention is the indicated treatment for such a case. It also must be resorted to in those cases of internal hernia of the newborn in which a knuckle of intestine is strangulated by the adhesions which sometimes anchor a Meckel's diverticulum to some other part of the abdominal contents. Hernias strangulated through the usual openings, umbilical, inguinal or femoral, seldom occur at a very early age.

Vomiting may be a symptom of increased *intracranial pressure* in the newborn. In such cases, it is explosive and refractory. In the earlier days of life, this type of vomiting is most often an indication of intracranial hemorrhage. Under these circumstances,

the vomiting will be accompanied by other and more striking signs of brain disturbance, such as changes in the reflexes, respiratory and circulatory slowing and irregularities, as well as by alterations in consciousness. Congenital hydrocephalus, the acute infective meningitides and encephalitides are sometimes causative.

The endocrine system as well as the brain may be responsible for vomiting of the most violent and intractable type. The best example of this is afforded by the emesis which follows **hemorrhage into the adrenals**. Here the vomiting continues almost without cessation and is accompanied by all the evidences of abdominal shock—pallor, dilated pupils, irregular respiration and disturbed pulse rate. Almost inevitably, death supervenes with rapidity and treatment is impossible.

In normal infants a certain amount of regurgitation of food may be considered physiologic. Given a vigorous baby and the *free flow of a copiously secreting breast*, overfilling of the infant's stomach is apt to occur, and as a result there will be a regurgitation of food. This may be accentuated by the presence of air which the baby has swallowed and retained in the stomach while lying in the recumbent position. This latter condition is easily remedied by picking the infant up and placing it in an upright position with its abdomen against the mother's shoulder; in this position the cardiac end of the stomach is highest. The overfilling is remedied, in the instance of an easily flowing breast, by allowing the infant to nurse but a few minutes at a time until it has taken the required amount. Another simple method is to have an ounce of milk expressed from the breast just before the child is allowed to nurse.

Qualitative abnormalities of secretion of the breast in the direction of overrichness or of poverty are especially fertile sources of emesis. In the first instance, the child may be given a dilute alkaline solution before nursing, and in the latter, supplementary feedings of artificial food with a time limit for nursing at the breast.

If the child is bottle-fed, an excess of either fat or carbohydrate may be the cause of vomiting. Carbohydrate vomiting is characterized by the ejection of watery, acetous-odored vomitus an hour to an hour and a half after feeding; the ejecta resulting from an excess of fat is creamy, yellowish and rancid. After a short period, during which nothing should be given the child other than dilute alkaline solutions sweetened with saccharine, adaptation

of formulas will usually meet the conditions. In the more persistent cases, stomach washing with a 1 per cent sodium bicarbonate solution may be needed before such a course is instituted. Meals which are too large, especially if these be coupled with overshort feeding intervals, may also be a cause of food ejection. On the other hand, many babies who vomit when they are given moderate amounts of food at very short intervals, may take considerably more when the time between the meals is increased to 4 hours.

Mechanical irritations of the fauces, such as occur when a child is given a nipple that is too long, or one in which the opening is so large that food reaches the throat with force and volume great enough to initiate a protective reflex, are productive of vomiting. Vomiting may also be brought about by the infant who sucks its fingers.

Toxic vomiting is the most frequent type encountered when dealing with infants from the second month to the end of the second year. This vomiting may vary in intensity from a slight regurgitation to a most explosive and prostrating ejection of all food and fluid. Almost all of the *acute infections* are ushered in by vomiting which, if it persists throughout their course, is of grave prognostic significance. With the *chronic infections,* tuberculosis especially, vomiting is a characteristic part of the clinical picture. *Uremia* and *peritonitis* are rare disorders during infancy, but when they occur, vomiting is a dominant symptom.

The irritating effect of drugs may be evidenced by emesis which sometimes persists because the character of the irritation is not recognized. Cough syrups containing a large amount of sugar, and such drugs as squill, ipecac and antimony may be causative even when given in minute doses, and the vomiting may continue after their withdrawal. Santonin, so popular as a vermifuge and so frequently administered by anxious mothers, is a common cause of vomiting. Calomel and other mercurials are badly tolerated by certain children; so also is hexamethylenamin. Close observation of the patient subject to such therapy may save the physician many an anxious moment. It may be of advantage to use glycerin rather than a syrup as a flavoring in medicines prescribed for the very young.

The syndrome known as **cyclical vomiting** may begin in late infancy; sometimes its onset is as early as the tenth month, but more often it comes on during the second year. The initial attack has all the characteristics of an acute duodenitis, and very often a carefully taken history will elicit that there has been

a persistent overfeeding of fat. Frequently these children are emotionally unstable and are of families some of whose members show constitutional inferiority of the nervous system. There is no question but that the vomiting is only a symptom, and it is seldom the first evidence to appear in the syndrome. Languor, fretfulness, irritability, headache, one or all, are apparent for some hours before the vomiting begins. Extreme constipation is almost always the rule, and the bowel movements are often characteristically foul-smelling and pale in color. The odor of acetone on the breath is usually to be observed in these cases.

Symptoms of infection of the nose and throat prodromal to the vomiting are so frequent that Eustace Smith and Sedgwick have incriminated the upper respiratory tract as the site of the infection which is etiological of the disease. Comby, on the other hand, believes that appendicitis is responsible for most if not for all of the attacks. It is probable that the symptom-complex can arise from various infecting agents. Sometimes the focus is in the upper respiratory tract; sometimes it is in the urinary tract; and sometimes in the intestine itself. It seems probable that a duodenitis is its most frequent precursor.

The symptom-complex is characterized by recurrent, intractable vomiting, a variable rise in temperature which may be excessive even to a hyperpyrexia, extreme prostration, constipation and thirst. As the hours pass, the picture becomes one of shock, and the symptoms of a true acidosis supervene. Restlessness and irritability give way to apathy and drowsiness. The navicular abdomen, hollow eyes and sunken cheeks, progressive diminution of urine, and thirst, all mark the loss of fluid, with the resulting concentration of the blood, which if unchecked will inevitably lead to a fatal issue. The detail of the treatment will vary according to the stage at which the physician encounters the attack. If he is fortunate enough to be called during the stage of restlessness and irritability, and if he is warned by the pallor about the mouth and nose and the highly flushed cheeks that toxemia is under way, the administration of a saline laxative together with a large saline enema may abort the disease. The same treatment may be effective if the vomiting has not persisted more than 24 hours.

We believe milk of magnesia to be the most effective purgative. It should be given in teaspoonful doses repeated every 20 minutes until from 10 to 15 teaspoonfuls have been given. It will be better retained if the dose is put in a dessertspoon containing

some shaved ice. Between doses the child should be fed small bits of cracked ice. The enema is prepared by adding 2 tablespoonfuls of sodium bicarbonate to 2 quarts of water. This is given through a size 16 to 18 English catheter 6 or 8 ounces at a time. Each injection is allowed to siphon off and only the last one is left within the gut. After the last dose of the laxative is given, fluid by mouth should be begun. It is our habit to use during the first 12 hours, 5 per cent sugar (either lactose, maltose or glucose) with 1 per cent or 2 per cent iced sodium bicarbonate solution. Some children tolerate this better if it is given in large doses, 3 or 4 ounces at a time. The first doses of it will be vomited, but if the ingestion be repeated promptly after each vomiting, the fluid washes out the stomach and very soon it begins to be retained. Certain other children, however, do better if the solution is given in teaspoonful doses every 5 to 10 minutes. In the second 12 hours of the treatment, high calorie orange juice (see Recipes) is substituted for the simple sugar solution and 24 hours later, solid food is allowed. The food best tolerated at this time has been found to be thin, thoroughly baked, dry toast with currant or loganberry jelly. In mild cases seen early, such a course of treatment will check vomiting and promptly restore the child to a normal condition.

It is essential that fluid be put promptly into the tissues in the severer cases where the stage of dehydration has begun, the toxemia is profound, and the child is drowsy and lethargic. The most rapid and effective way of doing this is by the intraperitoneal route; 100 to 250 c.c. of normal saline solution containing 1 to 3 minims of a 1 to 1,000 adrenalin solution is injected. (See chapter on Methods.) Fluid given in this manner may be excreted rapidly, and as we are dealing with a condition of concentration of blood proteins and lowered circulatory volume, it may be advantageous sometimes to use a colloid solution intravenously. If the child's fontanel is still open, the longitudinal sinus route should be chosen and the injection given, using the Goldbloom needle in the manner described in the chapter on Methods. If the fontanel be closed, the largest available vein and one of the simplest to use is the jugular. A hypertonic neutral solution of glucose, 15 per cent or 20 per cent, or the 7 per cent acacia solution of Hurwitz may be utilized as a fluid for injection. (See Methods.)

The only drug that can be recommended as of any value for the relief of symptoms in cyclical vomiting is opium. An injec-

tion of codein or of morphin given during the restless stage, in a dose proportionate to the child's weight will quiet the infant and bring it sleep; in the more toxic period, the same drugs will sometimes effect a prompt cessation of the vomiting.

The use of alkaline solutions by proctoclysis is often advised, but fluid administered in this way is less effective than when it is given by peritoneum or even by the subcutaneous route. (See chapter on Methods.) Sometimes proctoclysis cannot be carried out because of the child's extreme restlessness.

The prophylaxis of cyclical vomiting consists of searching out and remedying the infective process, if one can be found. Remembering the rôle played by duodenitis in the causation of the disorder, it is wise to limit fat in the food. Few fats are more damaging to such patients than cream and butter.

Contrary to the usual belief, sugar if it be given at meals, especially when it is cooked with fruits or diluted with water, is not deleterious but of distinct advantage as it encourages a saccharolytic flora in the intestine and tends to overcome constipation. The diet that we have found efficient in preventing the onset of the disease includes a daily ration of 1½ pints of high calorie orangeade (see Recipes) given in place of milk, which is withheld; well cooked, fine cereals, and other forms of easily cooked carbohydrates; fruits, cooked or raw; and a limited amount of meat.

It is hardly necessary to say that these patients should be given anesthetics only with the greatest caution, and that starvation should never be used as a therapeutic measure in dealing with them. Should it become imperative that such a child be anesthetized, a preliminary course of alkali will be in order.

Certain of the acute and subacute forms of **alimentary intoxication** that arise in infants, usually in the second half of the first year or the first half of the second year, are toxemias of graver import than those that lead to cyclical vomiting. Nevertheless, they are probably closely related in etiology, and for that reason the treatment detailed for the latter disease may be applied in exactly the same way for the relief of the vomiting in these intoxications. The group referred to rests upon an ultimate unknown etiology which, with Mellanby, we believe may prove to be the absorption of protein split products; and it is our opinion that a fertile source of such split products lies in high protein and low carbohydrate feeding, with the resultant establishment of a flora that elaborates toxic bodies related to the histamines and methyl-

amines. The body, in attempting to eliminate these toxins, sets up a vomiting or a diarrhea or both. Depletion and diminution of blood volume and concentration of blood protein follow, and the effective treatment must be directed toward remedying this pathology as well as toward increasing the elimination of toxins and the prevention of their formation. These aims are well met in the treatment outlined above.

Other cases of acute alimentary intoxication and vomiting arise in the course of saccharolytic fermentation as well as in the infective diarrheas, and the same picture in its greatest development occurs in the major stage of the bacillary dysenteries in which death is definitely hastened by dehydration. The treatment of vomiting as it occurs in this group of cases must depend on the restoration of the fluid balance through the checking of the diarrhea and by the measures enumerated in treating cyclical vomiting. It must never be forgotten that the Flexner-Shiga group of dysenteries are as amenable to treatment by their specific antitoxins as is diphtheria and that the diagnosis of dysentery calls for immediate injection of antidysentery serum.

Vomiting and Anorexia Subsequent to Weaning.—This group of cases of vomiting arises most often toward the end of the first year in patients who have just been weaned. The cases of this group have been considered as of nervous origin, but it is the writers' view that these are *toxic* in origin. The vomiting is always preceded by a period of anorexia, pallor and head sweating, and the clinical picture is generally one of subacute intoxication aggravated by self-enforced starvation. Stomach and colon lavage with alkaline solutions, and gavage constitute a form of treatment that is usually followed by a rapid amelioration of symptoms. In the less severe cases, spoon-feeding with thick formulas will meet the necessities of the case.

In the various types of vomiting at all ages, the *thick formula* is a most valuable therapeutic defense. Attention was first called to its value by McClure who recommended it as a feeding for the habitually vomiting baby—the infant whose condition has been variously described as pyloric spasm, mucous gastritis or as a vomiting of nervous origin. The vomiting in such cases is persistent, not copious and without pain; the ejecta consist of unaltered food mixed with some mucus. It is met with most commonly during the second and third three-month periods of the infant's life. It has one curious characteristic—the more liquid the food, the more persistent and profuse the vomiting. Be-

fore beginning the use of the thick formula (for its preparation see chapter on Formulas), it is well daily to wash out the stomach of the infant for a few days.

Obstructions in the alimentary tract are infrequent causes of vomiting. At levels higher than the pylorus, no obstruction is apt to be encountered although one or two cases are recorded of cardiospasm in infancy; and in vomiting occurring shortly after birth, congenital narrowing of the esophagus must be remembered.

Pylorospasm, uncomplicated, is commonly seen, although it is not present as often as it is diagnosed. However, as a complication of hypertrophy of the muscles at the pylorus (congenital pyloric stenosis) it becomes important. Cumulative, propulsive vomiting is an essential part of the clinical picture of obstruction at the pylorus in infants. The time of the onset of vomiting is also suggestive, as it is rare for such vomiting to be present before the end of the second week of life, and usually it does not begin before the end of the third week. The possibility of this pathological process evidenced by vomiting, makes it incumbent on the physician never to omit a scrutiny of the abdomen with the child completely undressed. Such visual examination is best made while the child is taking food. If the presence of a visible peristaltic wave is revealed, the diagnosis of obstructive muscular hypertrophy at the pylorus is more than probable. Confirmatory signs are the diminution of urine, scant bowel evacuations and persistent loss of weight. Of especial aid in arriving at a conclusion is the shield shape of the abdomen, wide and distended above and narrow and contracted below. One must not be misled by periods of amelioration with cessation of the vomiting, because spasm is a variable factor which at times may relax and allow some food to pass into the duodenum even when the hypertrophy of the circular pyloric muscle is extreme.

Surgical intervention is radical, simple and effective. The Ramstedt operation, first advocated by Fredet, can be done by the surgeon in a few minutes with the assurance that if the child is not extremely emaciated, recovery may be anticipated. The operation consists, briefly, of delivering the pyloric tumor, making an incision parallel with the long axis of the bowel, through the serous and muscular coats of the tumor down to the mucous membrane which must be left undamaged. Several modifications of procedures have been suggested; these call for the formation of flaps and for the protection of the pyloric wound

by stitching the omentum into it. Such procedures in our experience, are time consuming and of little value.

Certain important points of operative procedure may be dealt with here. The first is the surgeon's choice of the best location for the abdominal incision. We believe that the liver-protected incision as recommended by Butler is of distinct advantage. This incision is made just outside the right rectus muscle. The liver is so exposed that it can be raised with a retractor. The tumor will be found lying immediately beneath the incision where it can be delivered without extreme traction and with a minimum of shock. The great advantage of the incision at this site lies in the fact that when the operation is completed, the wound lies above the lower margin of the liver, and there is slight chance for a wound hernia. Before delivering the tumor, it is wise to pass a tube into the stomach and evacuate any gas that may be in that viscus. After raising the tumor, the operator's assistant should turn it over so it may be incised at a point on the posterior surface where the blood vessels are few and small. Great care must be taken that the duodenum is not nicked. It is a matter demanding considerable skill to divide the fibers of the lower end of the mass without injuring the duodenum. However, should the duodenum be nicked or the mucosa of the pylorus inadvertently entered, one or two fine catgut sutures will remedy the damage with no ill effects other than a somewhat less prompt cessation of vomiting after the operation. It goes without saying that before the operation the child's stomach should have been thoroughly washed out with an alkaline solution. This will prevent a postoperative diarrhea which sometimes is a serious matter for those patients in whom this precaution was neglected. Vomiting is less likely to persist after operation if the little patient is placed in a semi-erect position and propped up with pillows. (See Methods.)

The first postoperative feeding may be given within 4 hours and should consist of a 5 per cent lactose solution. Four hours later, in the case of breast-fed babies, a small feeding, half breast milk and half water, may be given, and in 24 hours the child may be given whole breast milk expressed from the mammæ and after 48 hours, nursing at the breast may be resumed. Artificially fed babies are usually first given a sugar solution and, after 12 hours, dilute condensed milk, 1-16. The concentration of the formula is slowly increased, until at the end of 3 or 4 days, it is possible to begin a rational formula of whole milk. Besides the adminis-

tration of water by mouth during the first 12 hours, it is the custom at the Children's Hospital in San Francisco to give water or glucose solution by the bowel. Gastroenterostomy has ceased to be a justified surgical procedure for the relief of pyloric obstruction with hypertrophy.

Unless it happens that the patient has come late to the physician the brilliant results which follow the feeding of *thick formulas* in some cases of undoubted plyoric stenosis, warrant the trial of this measure before a resort is made to operation. Certainly if the child is in good condition and not emaciated, no harm can be done by attempting this dietetic treatment, and in a large percentage of the cases it is certain that the symptoms will abate. The patient subjected to this course of treatment must be watched with the greatest care. Especially must the weight curve be observed. If a daily loss of 1 ounce or more persists over 4 or 5 days, the pyloric incision of Fredet must be undertaken. The details for the preparation of the thick formula will be found in the chapter on Formulas. There has been no really satisfactory explanation as to why the thick formula is tolerated in many of these cases, but the most plausible reason offered seems to be that the protective reflex of the pyloric muscle is inhibited by the colloidal character of the carbohydrate mass.

If the patient is at all dehydrated, which he is almost sure to be, it would be unreasonable to expect any dietetic or surgical treatment to succeed unless the fluid balance of the body is first reëstablished. Therefore, before beginning the dietetic treatment, it is wise to give one or two intraperitoneal or subcutaneous injections of normal saline solution or of Ringer's solution. While the thick feeding is being undertaken, meals of the cereal mixture may be alternated with feedings of water or of weak tea. It is also wise to inject 1 or 2 ounces of normal saline solution into the rectum every 4 hours for a day or two during the early days of the treatment.

The formula originally proposed by Sauer was made up with farina. We have found rice flour equally valuable, and it has the added advantage that it is less productive of diarrhea. Properly prepared, the food, when cold, should be as smooth as library paste and a little thicker; when warm, it becomes fluid enough just to flow. It is important that it should be kept warm while it is being fed. This can be readily managed by keeping the food in a bowl which stands in a basin of hot water. Some infants will accept the food from a spoon; for those who will not, we have

found it advantageous to use one of the large rubber breast nipples without a bottle. (See Methods.) After a few weeks' use, the preparation may be made a little thinner, and the child can then take it in the ordinary way from a bottle. It is good practice to divide the individual feedings into two bottles, one of which is kept in the basin of warm water while the baby nurses from the other. As the food cools and becomes thicker, the bottles are changed, and this alternation is continued until the entire feeding is taken.

The vomiting that indicates an *intestinal obstruction* is characteristic in that it begins by the rejection of the stomach contents and passes through the stage of biliary emesis, and if severe or neglected, eventuates in the vomiting of fecal material. Pain, constipation and abdominal shock are so much more the dominant symptoms of bowel obstruction, and the treatment of the vomiting is so entirely dependent on the removal of the obstruction, that a discussion of the management of this symptom, so frequent in the common obstructions such as *volvulus, intussusception and strangulated hernias,* will be reserved for another chapter.

The symptomatic vomiting of *appendicitis* and of *peritonitis* are amenable only to surgical treatment directed toward removing the cause.

Vomiting as a symptom of disturbances of the **central nervous system** has certain specific qualities. It is infrequently preceded by nausea; as a rule it is not exhausting; and it is accompanied by other signs of intracranial disease such as eyeground changes and headache. Uncomplicated by infection, the cause of such a state of affairs narrows down to **hydrocephalus** and the **brain tumors,** of which the commonest in babyhood is **glioma.** The treatment of the symptom is that of the disease. The corpus callosum puncture of Anton is applicable and sometimes curative when the vomiting is due to a hydrocephalus of the obstructive variety as may happen in the course of a brain tumor, congenital defect or in the wake of a meningitis.

The **vomiting that accompanies encephalitis, septic, specific or tuberculous meningitis** is as much evidence of toxemia as it is of mechanical irritation of the central nervous system. There is no treatment for the emesis apart from the management of the underlying disease, although as a temporary expedient nothing is more palliative than an injection of a small dose of morphin sulphate. Chloral by rectum may sometimes bring temporary relief.

Treatment of the symptom, vomiting, must be directed toward

a removal of its cause. But, apart from its cause, we have means of palliation and relief through stomach lavage and the administration of certain drugs, some of which are used as alkaline laxatives, some as local anesthetics, and some as sedatives to the nervous system. Of the first class, milk of magnesia is the best example; ½ per cent solution of phenol or ⅓ per cent solution of dilute hydrocyanic acid is illustrative of the second class; while in the third, opium and chloral in appropriate doses often give relief. But more effective than any other single measure is the reestablishment of blood volume by the introduction of water into the circulation. The best and simplest way to accomplish this state is to inject normal saline solution or Ringer's solution into the peritoneal cavity or under the skin.

Recently some clinicians have stated that the intraperitoneal injection of fluids is too dangerous a procedure for general use. An extended experience forces the writers to demur from this conclusion. Provided it is assured that the bladder is empty, that the stomach is not distended, that there is no peritoneal inflammation and that the technic is properly observed, no harm will follow the use of Ringer's solution or normal salt solution. The injection of other solutions is not without hazard. It is important always to have the tissues of the abdominal wall included between the thumb and finger and to feel the needle in position before making the final thrust into the peritoneal cavity. (See Methods.)

CHAPTER III

DIARRHEA

It must never be forgotten that diarrhea is a protective reaction—nature's attempt to rid the intestinal tract of deleterious substances. These substances may have been ingested; they may be the result of imperfect enzyme action or they may be the products of bacterial metabolism.

The normal breast-fed infant may have as many as 3 or 4 evacuations daily, while the artificially-fed rarely have more than 1 or 2 passages. The number and consistency of the evacuations varies with each child. The physical characteristics of the normal stool change with the type of feeding. Generally considered, however, the passages of a baby in good health should lie on the diaper as a homogeneous yellow, pasty mass—deep orange in the case of the breast-fed, lighter if the food given is artificially prepared. The odor should not be offensive; neither should it be highly redolent of acid.

Foul-smelling stools suggest the predominance of protein-splitting bacteria in the intestinal tract, a condition which may be remedied by diminishing the protein intake through curtailing the milk ration. While a putrid, foul-smelling stool always results from an excess of proteolytic organisms in the child's intestine, there are protein-splitting bacteria which do much clinical damage but whose products have little or no unpleasant odor.

When numerous foul-smelling stools are passed, milk should be entirely withdrawn at once. This period of complete withdrawal should be followed by another period of some weeks' duration, in which milk is to be used only in limited quantities. During this time the carbohydrate constituents of the diet should be increased sufficiently to meet the nutritional demands of the child.

On the other hand, when the evacuations are frequent, watery and excoriating, possessing the characteristic, but not unpleasant, odor of lactic acid, we are in the presence of a predominantly saccharolytic flora which can be and often is damaging to the patient. The logical remedy is to give such patients the food that will deprive the saccharolytic intestinal flora of those elements which can be readily converted into acids; therefore to increase proteins which

discourage the growth of this type of organism. The food that most effectively meets these demands is protein-milk. Because of its high efficiency in this class of case, it enjoys a widespread reputation. Unfortunately, it is often used without discrimination; and if it be fed to a child who is suffering from a diarrhea caused by the presence of inordinate numbers of proteolytic or putrefactive bacteria in the intestine, it can do only harm and sometimes irreparable damage. However, the signs and symptoms of the two types of food diarrhea are so clearly cut that the experienced practitioner will rarely mistake one for the other, even though he be denied the advantages of laboratory aids to diagnosis.

Hill of Boston has suggested a classification of diarrheas for the purpose of treatment. He divides them according to their etiology into mechanical, fermentative and infectious diarrheas. This classification can be made even more useful if a fourth group, the proteolytic, be added, and if the fermentative diarrheas be considered to be those which arise from a preponderance of carbohydrate splitting bacteria in the intestine. This brings the classification into accord with the etiological conceptions outlined in the last paragraph. We have, therefore, to deal with *mechanical, fermentative, proteolytic* and *infectious diarrheas.*

Mechanical diarrheas are rare during the first year but common during the second year of infancy. They always result from the ingestion of unsuitable food or of foreign bodies, such as dirt or paper, or from overfeeding with digestible food. Doubtless certain physical conditions of market milk allow massive coagulation in the stomach; these coagula, passing the pylorus, occasionally may act as foreign bodies and set up a mechanical diarrhea in young infants. Brennermann has laid stress on the value of boiling milk when such curds have appeared in the stools, and this procedure no doubt aids in bringing about changes that will prevent the appearance of diarrhea from this cause. The common practice of feeding yearling infants bits from the family table is to be deprecated because the food given may be causative of a mechanical diarrhea.

Such diarrheas are unaccompanied by fever, toxemia and prostration. The patient is readily restored to health by the institution of a bland diet which should be preceded by a brisk cathartic, such as castor oil, or calomel in divided doses. An insufficient dose of a cathartic but aggravates the condition. Castor oil in

full doses (2 to 3 drams at 1 year) is most effective; when it is given well warmed from a warm spoon, infants rarely object to it.

The nervous and emotional diarrheas, frequently encountered in older children and in adults, are so rare among infants that their treatment need not be discussed here.

Fermentative diarrhea constitutes a large proportion of summer diarrhea, a malady very prevalent and fatal in great cities. The general and undirected use of proprietary infant foods high in carbohydrate content is etiologic in many cases. It is probable, however, that proteolytic diarrhea is occasionally overlooked and treated as though it belonged in the fermentative group.

Close investigation of the history will reveal that diarrheas of fermentative type are rarely sudden in onset. Usually there will have been a prodromal period during which the child regurgitated a greater or smaller amount of sour, watery fluid about an hour before meals, and the attack of loose evacuations will have been preceded by transitory seizures of abdominal discomfort. Usually there has been an excoriation of the buttocks. The type of food has been one high in carbohydrate. Breast-milk, it must be remembered, is a food normally high in sugar. The child will be soiling from 5 to 20 diapers a day, the number varying with the severity of the attack. The stools are characteristic in that they are always copious, fluid, partly absorbed by the diaper, and of lactic acid odor; they tend to be green in color and to contain mucus mixed with semisolid fecal masses. Because the fluid part of the stool is readily absorbed by the diaper, it is easy to misjudge the volume of the evacuations. If a baby with fermentative diarrhea is put on a metabolism bed, the contrast between the amount of stool gathered by the bed pan with what is apparent when the same child is wearing a diaper, is most striking. There is no toxemia and rarely more than a little fever until the child begins to show the effects of dehydration which follow when loss of fluid from the bowel is prolonged.

Treatment in the earlier stages must be directed toward preventing this dehydration, and in the well developed cases toward combating it. The first and most essential steps are the withdrawal of carbohydrates and the provision of a high protein food which is readily available in the form of protein-milk. The method of its preparation will be found in the chapter of Formulas. Very satisfactory dried protein-milks are now available. These products meet all the essential requirements, are readily obtained, and are easily used.

Many authors deny the value of a preliminary purgation, but we cannot accept this view, for a forced evacuation of the bowel is found most helpful, especially in cases seen early. It is an excellent procedure to follow this evacuation by a copious high enema of a 5 per cent to 10 per cent sodium bicarbonate solution. If the lavage does nothing else, it neutralizes the acids which irritate the lower bowel; very often it also prevents proctitis—a distressing complication which may be the precursor of tenesmus.

During the first 12 hours of treatment, food should be given sparingly but water should be fed in quantity. Warm water is less likely to stimulate peristalsis than cold or hot water. In the second 12 hours, about one-third enough protein-milk should be given to meet the child's caloric demands. In this period, no sugar is to be added to the formula. However, on the following day an increase is to be made so that the protein-milk intake will suffice for one-half of the child's needs, and sugar must now be added. Dextrin in the form of corn syrup (brown) or malt-dextrin is the sugar of choice. One-third of an ounce to a 20 ounce mixture is the maximum amount that should be used at this stage, and from this time onward the proportion of protein-milk should be increased slowly, day by day, until the child's full nutritional needs are met. (That is to say, from 16 to 25 ounces of protein-milk with 5 per cent sugar each 24 hours.) To increase the sugar to the needed 5 per cent or 6 per cent will demand patience. In many instances it may be necessary to omit the sugar for a day, and then to return to the initial sugar content and work up to the tolerated maximum by small daily increases.

The treatment of the clinical aspects common to all acute diarrheas, whatever their etiology, will be considered at the end of this chapter.

Proteolytic diarrhea is characterized by frequent evacuations of yellowish or brownish stools, semisolid or liquid or mixed in consistency. Most often they have an exceedingly foul odor. There are, however, some patients whose stools are not offensive although they have a peculiar mousy odor; there are still others whose evacuations are almost odorless. These characteristics depend upon the nature of the proteolyzing organisms present in the stool but have little bearing on the mode of treatment. It is a safe assumption that diarrheic stools, which are definitely not acid nor the result of an infectious ileo-colitis, are proteolytic.

The severity of proteolytic diarrheas vary; some are very mild, and others are so severe that they threaten life. In the milder

cases, anorexia, pallor, sunken, deep-ringed eyes are evident; the temperature may be normal, subnormal or slightly increased; the pulse and respiration tend to be slow and irregular. The intermittence of the diarrhea is a prominent feature, and in many of the less severe cases the child will have 2 or 3 days of loose evacuations followed by a day or two of constipation. The distaste for food is profound. The child may even refuse to drink water, and as a result of the refusal the urine becomes concentrated and scanty. Most of these babies are restless and irritable, although occasionally one may be lethargic. The ingestion of milk is invariably followed by an increase of all the symptoms, and its withdrawal by a striking diminution in the number and volume of the stools and an amelioration of the toxic symptoms.

The clinical picture seen in the more severe cases varies only in intensity from that seen in the milder ones. The stools are more liquid and more frequent; sometimes 20 or 25 evacuations are passed in 24 hours. The appearance and odor of the stools are the same as in milder cases, but the toxic manifestations are of extreme severity, especially those whose incidence falls on the respiratory, circulatory and nervous systems. Restlessness and irritability are alarming and may give place to somnolence and even to coma in the full development of the disease. The pulse slows and becomes very irregular. Impending acidosis is made evident by the forced ventilation of the lungs with deep sighing and irregular breathing. Dehydration in these cases is usually less apparent than in the saccharolytic and infectious types of diarrhea, but it is not less profound in reality, nor is the need for injection of fluid less urgent.

The *treatment* for the toxemia and the dehydration is the same in this class as in the other forms of diarrhea and will be considered in a subsequent paragraph. The dietetic treatment is perfectly obvious—complete withdrawal of proteins for a short period. For 24 to 72 hours, nothing is to be given except a 10 per cent sugar solution or a dextrin decoction. Ordinary cane sugar (saccharose) is not effective; milk sugar (lactose) is by far the most desirable form in which sugar can be given. The best routine is to initiate the treatment with lactose solution (2½ ounces of lactose to 30 ounces of water) in divided doses every 2 hours. At the same time, especially if the child shows evidence of a mild dehydration, normal salt solution should be given by mouth, 1 to 2 ounces at a time. In those cases in which toxemia is well developed or in which dehydration is advanced, 100 to 300 c.c.

of normal salt solution should be given intraperitoneally; this may be repeated after 12 and 24 hours if needed. After 24 hours of exclusive lactose solution feeding, cereal gruels may be added to the diet, and the proportion of lactose in the food reduced to 5 per cent. Experience approves a farina or oatmeal gruel made by cooking 1½ ounces of the cereal and 1½ ounces of lactose in 30 ounces of water. These ingredients should be boiled together for 1 hour and then strained. If desired, this gruel may be slightly sweetened with saccharose.

Exclusive carbohydrate feeding is maintained for from 1 to 2 weeks; milk is then gradually added to the diet. In order to limit the amount of protein added and to increase the child's tolerance for milk, it is wise to begin by using top-milk in very small quantities. The initial amount to be given is 1 ounce of the top 5 ounces of a quart of milk added to each 20 ounce mixture. This adds 1 per cent of fat. After a few days, a second ounce, and some days later a third is introduced. This 3 per cent concentration provides a food as high in fat as most patients will tolerate well. From this point on, instead of the top 5 ounces, it is well to use the top 8 or top 16, increasing the proportion of the milk in the mixture so that in 6 or 8 weeks the child is getting the normal daily ration of 1½ ounces of milk per pound.

The conception that protein splitting bacteria found in the intestine are etiological in any of the nutritional or digestive disturbances of infancy is unacceptable to some able pediatricians. It is the belief of the writers, however, that evidence both clinical and bacteriological makes this conception a useful working hypothesis. The technical complexities of the problem and the limited amount of material studied render recent bacteriological observations entirely inconclusive; so that for the present at least we must be guided by our clinical experiences.

The **infectious diarrheas,** dysentery and ileocolitis are generalized bacteriemic infections. Their intestinal symptoms are the result of the invasion of the intestinal walls by bacteria whose life processes produce destruction of the mucous membrane. The evidence of this destruction appears in the bowel evacuations as blood, pus, epithelium and masses of necrotic tissue. While it is an exaggeration to say that every stool containing pus and altered blood is the result of a true infection of the intestine, it is a wise clinical rule to treat a patient with such symptoms as though the infection had been proved before waiting for laboratory confirmation.

The cases which are the most severe and which most tax the

skill of the physician are those following an invasion by the dysentery group of bacilli. The Flexner, Shiga or Hiss-Russell types of organism may be etiologic. The Shiga bacillus, which produces both a neurotoxin and an enterotoxin, is the most to be feared because of the profound toxicity of its growth products; but the other bacteria of the dysentery group lack little in poisonous properties.

It is in the treatment of the diarrheas of this etiology that the most brilliant results follow the administration of specific sera.

In the *treatment* of dysentery of the Flexner, Shiga or Hiss-Russell types, the early and ample intravenous injection of a polyvalent dysentery serum is specific. The dose must be sufficiently large to meet the clinical indications. A total of 200 c.c. or even more should be given in doses of 50 c.c. every 5 or 6 hours, or until there is improvement in the child's condition. It is well to dilute the serum with glucose solution and to give it intravenously in order to combat the dehydration.

In the 1919 epidemic in Portland, Oregon, in which Hiss-Russell dysentery bacillus was the causative organism, Karl Meyer, directing the bacterial aspects of the attack, was able to check the toxemia with great promptness through the use of Flexner's polyvalent serum. During this epidemic, Meyer observed that many of these dysentery patients died some days after the toxic symptoms referable to the action of the dysentery bacillus had been overcome by the use of serum. Although the stools had abated in frequency and in volume and had lost their purulent and bloody aspect, and in spite of the fact that the acidosis had been combated and the water balance maintained by the attending physicians, these cases went on to dissolution. The patients died with such great abdominal distention that in many instances respiratory and circulatory embarrassment occurred.

The explanation for this state of affairs was found and a cure effected when it was recognized that the trouble lay in the great amount of intestinal detritus, the result of the activity of the dysentery bacillus. This detritus, consisting of shed epithelium, pus and sloughs, presented an ideal feeding ground for the enormous numbers of facultative spore-bearers, especially B. Welchii and butyric acid producers—organisms so often found in the stool of the dysenteric patient. It will be recalled that facultative organisms are those that thrive either on carbohydrate or protein media. The observers found that, when it was possible to swing these concomitant flora from a proteolytic to a fermen-

tative balance, the patients rapidly improved and the chronic postdysenteric phases of diarrhea and malnutrition were aborted.

In this connection it was discovered that an attempt to shift the flora immediately by a too rapid change in the pabulum might result in a fatality. Kendall observed that dysentery bacilli grown on media rich in carbohydrates are less toxic than those grown on a protein medium. Under the influence of this idea, the attempt had been made to treat some of these patients dietetically. Protein was excluded and sugar solution only was given, with results that were sometimes disastrous. Under such treatment, enormous quantities of gas were produced in the gut, and butyric acid in quantity sufficient to be irritating appeared; so that a few days after the abatement of the dysentery, a secondary diarrhea resulted, a diarrhea accompanied by a rapidly ensuing toxemia which led to the death of the patient.

Karl Meyer's explanation, and his treatment of this phase of the condition rested upon the fact that spore-bearing, facultative groups of organisms occur in the intestine in two main developmental stages, the spore-bearing and the active. He conceived that in the presence of the detritus in the dysenteric bowel, these organisms become highly active and take on a vigorous growth, producing large quantities of low grade poison, to which the body is more susceptible because of its lowered resistance that has resulted from the dysentery. These organisms are in antagonism to the aciduric group (B. acidophilus and B. bifidus), so that the growth of these latter is discouraged almost to the point of disappearance. If at this time the child is given a food so high in sugar that some of the carbohydrate reaches the ileum and upper colon unchanged then the vigorously growing facultative organisms seize upon the sugar and break it down, with the result that there is an enormous production of gas by B. Welchii and some of the other spore-bearers, and the few aciduric organisms present are unable to establish themselves as they would were the spore-bearers not predominant.

In order to meet this newly discovered situation, it was conceived that could the spore-bearers be forced back to the resting or spore stage, in which they have difficulty in utilizing carbohydrates, the essential saccharolytic flora might be encouraged to grow vigorously. After due consideration it was accepted that the time-honored though often abused method of starvation was really of use in this type of diarrhea, because it achieved just this purpose; and that the equally time-honored use of castor

oil to sweep out the bowel is valuable. A starvation period of 12 to 24 hours proved to be sufficient to inhibit the activities of the spore-bearers, and one or two ample doses of castor oil before and after this period, added to the effectiveness of the treatment. During such treatment, the free use of fluid is essential; it must be given by mouth, and subcutaneously or intraperitoneally if need be. Barley water by mouth, and plain water or normal salt solution by injection may be used.

While a period of limited starvation and purgation is desirable, to continue it overlong is a procedure fraught with danger. As dysentery patients must be supported, carbohydrates are to be fed in increasing amounts so that the optimum is reached in about four days. In this way the facultative spore-bearing bacteria are still held in abeyance, while the lactic acid producers that combat them are mobilized in full force. Following initiation of this plan, the fatalities of dysentery have been markedly reduced, and the apparent intensity of the intoxication diminished.

The *typhoid, paratyphoid* and *paracolon* groups of organisms are responsible for a small number of infective diarrheas. Unfortunately we have no specific serum with which to combat the toxins produced by these microorganisms. In general, the treatment for any of these infections is identical with that outlined for the treatment of dysenteric diarrheas except in the matter of specific serums. However, dehydration is not so much a feature, and injection of fluid is not so essential a part of treatment; although it is probable that the course of the typhoid and paratyphoid groups is favorably influenced by the intraperitoneal injection of fluid, especially in those cases in which toxemia is profound and the sensorium is markedly depressed.

The virtue of the high calorie diet originally recommended by Coleman undoubtedly rests partly upon its ability to inhibit overgrowth of concomitant proteolytic flora. The management of such cases is essentially directed to the nutrition of the patient to which the high calorie diet contributes efficiently. For the rest, the symptoms and complications are to be met by appropriate treatment as they arise.

Occasionally B. pyocyaneus causes a persistent diarrhea, difficult to differentiate from the diarrhea of the paracolon group. The treatment is essentially the same as that outlined above.

The diarrheas of a *streptococcus* origin are infrequent, although it is common to find the streptococcus in the evacuations of infants suffering from diarrhea. In most cases, these are but casual

invaders, and many of them are of the nonpathogenic types. When the streptococcus is truly etiologic of a diarrhea, the cases occur in groups; and almost inevitably, careful search will incriminate some article of food which has been contaminated with pathologic streptococci and ingested by the members of the group. The food most often responsible for these epidemics is milk, but other foods may be carriers and direct infection from case to case has been frequently traced.

The characteristics of a true streptococcus diarrhea are the suddenness of the onset, often a chill, high temperature, excessive toxicity and a rapid course to death or recovery. The stools are neither very numerous nor copious, (8 or 10 a day as a rule). Sloughs are almost unknown in the evacuations; blood when it occurs is in small quantities. Often, however, the stools contain serum which is blood streaked and stiffens on the diaper. Prostration and collapse are frequent symptoms which may occur early. In most of these cases, the diarrhea is but one phase of a streptococcic septicemia.

While a polyvalent streptococcic serum has nothing like the specific effect of the Flexner's polyvalent serum for dysentery, it is nevertheless of great value and should always be used. It is valueless unless it is used in large quantities frequently repeated. It is essential that the nervous and toxic symptoms of streptococcic diarrhea should be combated with hydrotherapeutic measures and by the use of the mustard pack. (For technic see the chapter on Methods.)

General Scheme of Management

When we are confronted with a case of diarrhea, it is wise to review all the possibilities of treatment. To accomplish this with an economy of time and effort, it is desirable to have some plan in mind as a guide. A mild attack of diarrhea may become severe with such rapidity that it is the part of wisdom to be prepared for all eventualities.

The first thing to determine is the etiologic category into which the case falls. Once this is decided, it is well to consider the following questions:

1. Is a period of starvation essential?
2. What foods may be used with advantage?
3. How are the water needs of the child to be met?
4. How shall body temperature be controlled?
5. How can rest be assured the patient?

6. What drugs are of value in the treatment of this patient?
7. How can we prevent the spread of the infection?

1. **Is a period of starvation essential?**

Certainly, if there has been no previous starvation, either voluntary or enforced, it is good practice to withhold food for 12 hours; sometimes 24 hours' deprivation may be imposed with advantage. The use of fluid, however, is essential from the first. This may be given in the form of plain water, barley water, normal saline solution, or weak tea; the latter is of great value in those cases in which stimulation is needed. A short period of starvation, if fluid is freely given, can never harm the patient; but undoubtedly many patients have suffered through long-continued food deprivation.

2. **What foods may be used with advantage?**

The answer to this question will depend on the type of intestinal flora. Some of the cases seen in this country are proteolytic diarrheas. When the stools lack the characteristic excoriating acidity and sour odor found when saccharolytic bacteria are predominant, no harm can be done by passing from the cereal water that has been used during the starvation period to a 5 per cent gruel of the same cereal. Nor in the absence of these stool signs is there likely to be any damage done by the addition of the sugars (lactose or malt sugar) and dextrins to the cereal gruels in amounts at first not to exceed 1 level tablespoonful to a 20 ounce mixture. These amounts may be increased day by day in the absence of untoward symptoms, such as high gas production or the appearance of excoriations on the buttocks of children who are well cared for.

If the stools become more solid, less frequent and less foul, as is usually the case, recourse may be had to malt-soup. (See chapter on Formulas.) Care must be taken to keep the milk used in the first feedings of malt-soup down to the minimum. More than 1 ounce of milk to a 20 ounce mixture should not be used in the beginning. It is well at first to take this ounce from the top of a quart of milk on which the cream has risen. If this mixture agrees, a second ounce of the same strength of top-milk may be added, replacing an ounce of water. The following day a third ounce may be substituted; but for fear of having too much fat in the mixture, this third day's feeding should be taken from the top 10 ounces of a quart of milk. Daily 1 or 2 ounces of milk may

be substituted in the mixture until the proper proportion of milk (1½ ounces per pound per day) is reached. As more milk is added, the lower levels of top-milk may be used; so that if ⅓ of the mixture is milk, the top half of the quart is used to provide it. When the proportion of milk reaches ½, well shaken whole milk should be employed in the preparation of the food.

Raw milk should never be used in formulas for infants under treatment for diarrhea. Boiling the milk renders it less readily attacked by bacteria in addition to changing its physical and chemical properties in a way that makes it more easily digestible.

Children in later infancy who are attacked by *proteolytic diarrhea* are more comfortable and return to health more rapidly if milk is excluded from their dietary altogether. These little ones are best fed on cereals, toast with fruit jellies, simple puddings of cornstarch and gelatin, and the lighter broths, such as chicken and veal, thickened with barley, sago, tapioca, cornstarch, arrowroot or other easily digestible starches. Potato, because of the peculiar character of its starch capsule, is difficult to digest properly and is one of the last foods to be added to the dietary of a child who has suffered from a diarrhea. It is very often responsible for mild relapses.

In this type of diarrhea, cooked fruit purées (passed through a fine sieve) need not be withheld, although there is a great prejudice against fruits as part of the diet in diarrhea. These purées are especially valuable in the proteolytic types of diarrhea and in the later stages of the infectious dysenteries because they can carry considerable additions of lactose or glucose in the form of milk sugar or corn syrup. These sugars are especially valuable under such circumstances, for they supply energy, act as diuretics, and discourage the establishment of a proteolytic flora in the gut.

In *saccharolytic diarrhea,* when the stools are characteristically acid, excoriating and acetous in odor, whether or not they contain much gas, the dietetic treatment is essentially the opposite to the method of procedure just outlined as effective in the management of putrid diarrheas.

Here there is no question but that the sugar-splitting bacteria are producing the conditions that result in excessive evacuations and that the further use of carbohydrates can but aggravate the trouble. It is one merit of the Finkelstein school that they presented protein-milk to the world. This is a food almost specifically ameliorative of this type of diarrhea. (See Formulas.)

The preparation is a matter of some difficulty and this food is rarely properly prepared outside the diet kitchen of a hospital or in a milk laboratory. Therefore, the presence in the market of a dried form of protein-milk, prepared simply by the addition of water, adds an invaluable dietetic weapon to our resources.

It must be remembered that a short period of starvation is essential before the initiation of this treatment, and when breaking the fast it is imperative that too large quantities of food should not be given at first. If protein-milk itself is used, it is well to begin with not more than 5 ounces in a 20 ounce mixture; as the child's tolerance becomes established, the amounts are increased up to 15 ounces in a 20 ounce mixture, or it may even be given undiluted.

Early in his advocacy of protein-milk, Finkelstein frowned on any sugar additions to the formula, but he soon learned that sugar deprivation over an extended period was fatal to his babies and he even published an article in which he called attention to the need for carbohydrate additions to the preparation. Unfortunately a number of his pupils failed to follow him in his change of ideas. There is no doubt, however, that in taking this ground, Finkelstein was entirely justified, and the successful use of protein-milk depends upon the coincident use of small but daily increasing amounts of sugar, preferably malt-dextrin mixtures or corn syrup. Otherwise the glycogen reserve of the body becomes impaired and the nutrition and resistance rapidly fail.

In practice then, we use protein-milk without sugar for not more than 24 hours. Then malt-dextrin, or corn syrup should be added in the proportion of ½ of 1 per cent (1 teaspoonful to a 20 ounce mixture), increasing 1 teaspoonful every second day until 6 teaspoonfuls have been added to the 20 ounce mixture. This brings the final sugar concentration up to 3 per cent, which is about as high a percentage as can be used with safety while the evacuations are loose or frequent.

The success of the treatment is evidenced by the decreasing number of stools which acquire the characteristic soapy appearance, pale and hard, with an odor of fatty acids. The first sign of the overuse of protein-milk is a change from an acid to a foul odor in the stools. Such a change is a valuable warning that the carbohydrate content of the food should be increased. It matters little whether one of the sugars or a dextrin is used for this purpose. The popularity of many of the proprietary foods depends upon the large amount of the latter carbohydrate in

their composition. One of these, such as Imperial Granum, may be used; but if there is any prejudice against the use of a proprietary food, the same effect may be had by using flour-ball, baked flour or thoroughly browned toast crumbs ground fine in a coffee mill. Even thoroughly boiled starch assumes a colloidal form which has a definite place as an adjuvant. Much as we may condemn the methods of exploitation of the proprietary food manufacturers, convenience and availability are on the side of their products and sometimes the exigencies of circumstances will force their use. If they are used with knowledge of their composition and with care and discrimination, they may be of great value to the physician who has to treat infants suffering from diarrhea.

With the diminution in the number of stools, normal formulas are to be substituted by replacing one bottle a day of the protein-milk with one of a boiled-milk mixture. It is best in the beginning to make the proportion from ½ to ⅔ skim-milk. As improvement in the symptoms occurs, more feedings of the boiled skim-milk mixture may be substituted for the protein-milk until the child is taking a rational formula for its age. After the first day, less cream is removed from the bottle and the whole milk mixture is reached by gradual stages. It is often necessary, however, to return temporarily to the use of protein-milk mixtures because it may be difficult to re-educate the intestine to tolerate dairy milk mixtures.

In spite of the inferences to be drawn from the *in vitro* experiments of Kendall, the type of feeding has little or no effect on the character of the dysentery bacillus in the intestinal tissues, and the attack on this organism must depend upon the use of appropriate serum therapy. Discussion in the paragraph on etiology will illustrate the writers' belief in the importance of a concomitant flora as one of the factors producing a grave and often fatal complication during the course of an otherwise mild dysentery. Confronted with one of these infections as evidenced by frequent, hemorrhagic, purulent stools and a tendency to rapid dehydration, our effort should be to inhibit the growth of the spore-bearing saprophytes, especially B. Welchii, even in the absence of bacteriologic proof. To do this, a period of partial starvation of two days is advisable. During the first day, if the child has not already been starved, food deprivation should be positive and nothing should be given but weak tea, barley water or albumin water. On the second day, broth with egg white may be

given, or if the child is very weak or has already been subject to a period of starvation, a dilution of protein-milk (1 to 3 in water sweetened with saccharin) may be allowed. This day's ration should not exceed 6 to 10 ounces of protein-milk without sugar. The following day 8 to 12 ounces of protein-milk may be used, together with ½ of 1 per cent dextrin (dextri-maltose, flour-ball, corn syrup) and 2 per cent barley decoction. Day by day, with the improving condition of the child, an increase in the amount of the carbohydrate should be made until a 5 per cent sugar and a 3 per cent to 5 per cent cereal or dextrin gruel is being used, together with a moderate amount of protein-milk, the greatest quantity of which should not exceed 16 ounces in a day.

For older infants in the convalescing stage of the disease sago, barley, arrowroot, cornstarch, and the other cereals may be made into simple gruels, porridges and puddings to which egg yolk may be added for its nutritive value. Alcohol in the form of brandy, whiskey or simple elixir may be used for its caloric value and may be considered a food rather than a drug. Great care must be taken to give it in sufficient dilution, ½ per cent to 1 per cent in water is most useful and the daily intake must be limited according to the weight of the patient.

3. **How are the water needs of the child to be met?**

It must be recognized that the supply of an adequate amount of fluid is the sheet anchor of treatment in the diarrheas. Children have been allowed to suffer for the lack of water because the ingestion of water by mouth initiates peristalsis and often increases the number of stools. We must not allow this fact to intimidate us. Very often by giving the fluid at body temperature or by incorporating in it an infusion of tea we can minimize this tendency. In those cases in which the rapid and frequent evacuations and loss of fluid by the bowel are so threatening that they force us to withhold water by mouth, we must be prepared to inject fluid under the skin or into the peritoneum. If the technic given in the chapter on Methods, is used in giving fluid intraperitoneally, no trouble will follow. Care must be taken to maintain the fluid at a proper temperature, for the child may be easily chilled. In cases of collapse with low temperature, there is a great advantage in having the water enter the peritoneum at a temperature not less than 116° or 118°. Amounts of normal salt solution varying with the size of the child, from

100 c.c. to 300 c.c., can be given at 10 or 12 hour intervals until the dehydration has been overcome. The advantage of this method over the subcutaneous is that it is less painful, that it produces less shock and there is a better retention in the blood stream.

When the subcutaneous route is chosen, the site for the injection is either in the axillæ or in the abdominal wall. If a needle sufficiently large is chosen, and a good technic acquired, 100 c.c. easily can be put into each axilla without damage to the patient, and as much as 150 c.c. will be tolerated in the subcutaneous tissues of the abdomen on each side of the median line. The volume accepted will depend largely on the degree of wasting and dehydration exhibited by the child. The technic for hypodermoclysis will be found in the chapter on Methods.

In cases in which vomiting is a feature, gastric lavage is indicated. In the more severe cases even where there is no vomiting, a stomach washing daily for 2 or 3 days is of great value, not only because it may remove toxins excreted into the stomach, but also because it is desirable to leave a considerable quantity of water or dilute alkaline solution in the viscus. Water given in this way does not seem to stimulate peristalsis to the same degree as when it is swallowed. Either 0.8 per cent salt solution or 2 per cent sodium bicarbonate solution may be used according to the plan outlined in the chapter on Methods.

The use of proctoclysis is out of the question in such cases because of the irritability of the bowel.

4. How shall the temperature be controlled?

Fever control is of great importance when we are dealing with infectious diarrhea; and the maintenance of a cool surrounding atmosphere during hot weather is of equal importance in the treatment of the noninfectious types.

When the patient's temperature ranges above 103°, the use of a tepid bath or cool pack may be a life-saving measure. If there be cyanosis or collapse as well as a high temperature, the mustard pack is preferable to the simple water pack. Properly given, it will reduce temperature because of the dilatation it induces in the vessels of the skin and the perspiration and consequent evaporation it produces. At the same time, it stimulates the sensorium and increases the power of the circulation and the ventilation of the lungs. It is of equal if not of greater value in those cases of infectious diarrhea with low temperature and collapse. Details for its preparation and use will be found in the

chapter on Methods. In the mild cases, tepid, hot or mustard baths may be substituted for the pack, but properly given the pack is to be preferred.

The use of enemas may contribute to temperature reduction; however, no method of procedure is more overdone than this. If given cold, they may cause such rapid loss of heat that the patient collapses. It is never wise to give a cold enema, and in but few cases is it desirable to give an enema daily for longer than a week.

To maintain a mild surrounding temperature is as important as to keep the child's fever at a low level. This may be best accomplished by choosing a large, well-ventilated room for the patient and by the use of electric fans, or failing these, in hot climates by the use of rapid evaporation of water on the olla principle. The chapter on Methods gives a description of an apparatus based on this idea.

For the control of fever in this group of diseases, drugs are unnecessary and often damaging.

5. How can rest be assured the patient?

No single therapeutic measure, excepting maintenance of water balance, is more effective in the treatment of diarrhea than the obtaining of tranquil repose for the child. In the presence of an excitable family, this will sometimes tax the resource of the physician. Whenever possible, a trained attendant should be employed. Choice of a nurse will depend on circumstances, but one who is experienced in the care of sick infants may turn the balance between success and failure in the treatment. The exclusion of visitors is important even to a young infant. The forbidding of any handling of the child by another than the nurse, or by the mother at feeding time if breast feeding is maintained, is imperative. The use of packs for their tranquilizing effect on the nervous system is advisable. The judicious selection of one of the many hypnotic or sedative drugs may be indicated; particularly opium and in some cases chloral may be found very useful.

6. What drugs are of value in the treatment of diarrhea?

Innumerable drugs are used, most often unwisely, in the struggle against diarrhea. Frequently they are not only useless but actually harmful in their effect and they may divert attention from more important therapeutic measures. The astrin-

gents are of little or no value in the acute stages of diarrhea, and their use might well be abandoned.

There are a few drugs which have a high reputation and certainly are of value in the subacute stages of the disease. These act as inhibitors of bacterial growth in the lumen and on the intestinal wall. They must be given in full doses and with some frequency. In this group are included bismuth, bolus alba, and animal charcoal. The most commonly used of these is bismuth, and it is best prescribed in the form of the subcarbonate in the subacute stages of the disease. A reliable product must be chosen, as many commercial samples are contaminated with arsenic. The drug should not be used in the more acute stages of diarrhea. The minimum dose for a baby should be 15 grains at 3 hour intervals, and the maximum 40 grains at the same intervals. These powders can be added to the food and are well taken. Bolus alba can be substituted for the bismuth in somewhat larger doses. It is rarely effective in a dose less than from 40 grains to a dram every 2 or 3 hours. It is a very effective aid to the dietetic treatment in the saccharolytic diarrheas and is apparently of some value in the middle and later stages of the infectious diarrheas. It is especially recommended by Austrian pediatrists for that stage of the infectious diarrheas in which bismuth is useful, that is, when the sloughs have disappeared from the stools and the intestinal ulcers have begun to heal.

Mercury, silver, salol and the salicylates by mouth as intestinal antiseptics have little worth. The use of calomel for the initial purge in the treatment of diarrheas is time-honored and undoubtedly it is effective, but castor oil is to be preferred. It should be used in full doses, certainly not less than 2 drams for babies 6 to 10 months old and 3 to 5 drams during the second year. Children take it very well, especially if it is warmed and given from a warm spoon. The continued use of calomel as an intestinal antiseptic which was in vogue a few years ago is not justified and it may be productive of great harm.

The French pediatrists have recommended the use of hypodermic injections of emetin for the bacillary dysenteries. They state it is as effective in these types as it is in the amebic group. The writers have used it with gratifying results, especially in the nonspecific infectious diarrheas. Its use, however, is merely as an adjunct to the dietary and symptomatic treatment. A single daily injection is made in dosage appropriate to the child's weight.

The use of opium by mouth as a remedy in diarrhea has been vigorously opposed, but the value of the drug when properly given is attested by the fact that the more experienced a physician is, the more convinced he is of its usefulness. On the other hand, without question this drug has been more abused than any other. No reasonable practitioner would prescribe morphin or any other opium derivitive for a diarrhea during the stage of intoxication and high fever; but it remains the most potent weapon we have to check the excessive number of bowel movements in the subacute stage of the disease. It is invaluable to procure rest and to soothe the overirritated nervous system of the child. When used, the drug should never be added to a diarrhea mixture. Absolute control, through accurate dosage, must be maintained. It must not be forgotten that certain infants are exceptionally susceptible to its effects; and yet to obtain an effective result, a full dose is necessary. It is wise therefore, when beginning to use the drug, to give one of the less potent derivatives, such as codein, in order to determine the child's tolerance.

If the patient is found to have a reasonable tolerance, then morphin may be used in appropriate doses. Weight is the best indicator of proper dosage. A child of 15 pounds will usually tolerate 1/10 of the adult dose. In using codein, it is our practice to use a ½ of 1 per cent solution and to give a dose by mouth proportionate to the weight of the child. If this is not effective in calming the baby or in checking the excessive peristalsis during the subacute stages, and the infant shows no evidence of narcotism, we change to a $\frac{1}{10}$ of 1 per cent solution of morphin, measuring the dose to be given by the criterion of weight. Usually a child of 6 months will take $\frac{1}{20}$ to $\frac{1}{24}$ of a grain of codein or $\frac{1}{30}$ to $\frac{1}{40}$ grain of morphin, repeated not more than once or twice in 24 hours. There is an old and valuable rule that no patient should be wakened to receive a medicine containing a narcotic. If this rule is followed and opium is not given in mixtures, no bad results can follow doses of the size mentioned.

Whether the diarrhea is of an infectious or a noninfectious origin, the circulatory and nervous complications are a constant source of danger to the child. Under these circumstances there is no drug so valuable as camphor which should be given alone. The average dose for a 15 pound child is 5 minims of a 10 per cent solution of camphor in sterile olive oil given hypodermically. The dose may be repeated as often as every 2 hours when the pa-

tient is severely prostrated, but in general, 1 to 4 doses daily will be sufficient.

When abdominal pain is a complication of a diarrhea, atropin is a drug of great value. Babies sometimes tolerate surprisingly large amounts of the drug. The injection of $\frac{1}{800}$ of a grain for a 15 pound baby will often check a tenesmus or the abdominal cramps that accompany a diarrhea. It is also effective at times in assuaging the irritability of the gut and in reducing the number of evacuations.

Cocain in $\frac{1}{10}$ of 1 per cent solution has been recommended and is useful in checking the frequency of the stools under the same circumstances in which opium is indicated. However, it is a dangerous drug and it must be used with caution.

A ¼ of 1 per cent solution of aluminum acetate used as an enema is very valuable in cases of diarrhea of saccharolytic origin, especially those in which there is much mucus in the evacuations.

Tenesmus is best treated by enemas of starch and opium or of a suspension of bismuth subcarbonate in mucilage of acacia. A solution of cocain, $\frac{1}{20}$ of 1 per cent, used locally in the rectum has been recommended to relieve tenesmus and is sometimes of real value.

The specific serums, of course, must be used in all appropriate cases. They are given intravenously, and it is desirable to give them in high dilution. They should be injected slowly in doses apportioned to the weight of the child. (See Methods.)

7. How can we prevent the spread of the infection?

It is most important that as many infants as possible be breast-fed. Once a case of diarrhea develops, it is essential to initiate a rigid prophylaxis immediately. The attendant, trained or untrained, must be taught to observe precautions as scrupulously as though the case were surgical. Basins for washing and sterilizing the hands must be maintained close to the patient's bed. The attendant's least contact with the person of the infant should be followed by an ablution with soap and water, preliminary to rinsing the hands in a ½ per cent saponified cresol solution. It is well to remember that soap solution is one of the best bactericidal agents obtainable. After evacuations, the washing of the soiled buttocks with soap and water, followed by prompt drying, is a valuable means of preventing the speading of the disease through contamination.

The immediate change and the proper care of bedding after

it has been soiled must be scrupulously observed. The simplest means of dealing with soiled bedding and clothing is to have a large wash boiler containing a solution of cresol ready for their reception. When the boiler is half filled with bed linen, its contents may be boiled for 20 minutes. This minimizes the needed handling and guards the soiled articles from contact with flies and other insects. Pieces of old linen or several thicknesses of cheese cloth should be used to receive the stools and these are to be burned immediately after they are removed from contact with the child. A diaper made of gauze, cotton, or cotton fiber and absorbent paper which may be burned when soiled is used very successfully at the Babies' Dispensary in Philadelphia. It has an economic advantage as well as a hygienic one.

Screened windows are of great advantage. Failing these, it is essential to protect the patient's bed by a canopy of fine mosquito netting.

A pan of soapy water should be standing near, into which all used dishes should be immediately dropped; later the pan should be set on the stove and the contents boiled. All unused food should be destroyed.

It is important that the attendants wear washable dresses or slip-over gowns which should not be worn out of the room. Appropriate precautions should be taken in their laundering. The isolation of the patient suffering from diarrhea should be as positive as it would be if the disease were scarlet fever, diphtheria or any other of the highly communicable disorders.

The breast-fed baby fortunately is well protected from diarrhea. The severe diarrheas which attack a nursing infant are most often of the infective type. The child may be infected from some one else in the family or from a child in a neighboring home; very often the mother may be the infector. It is not necessary that she should have a severe diarrhea at the time; she may have acquired immunity and have become a carrier—a source of danger to her infant and to others in close contact with her.

In the absence of a household epidemic, it will be found in the majority of cases of diarrhea affecting the breast-fed that the child is receiving supplementary feedings, often against the physician's orders and unknown to him.

In all details except the feeding itself, the *treatment of infectious diarrheas in the breast-fed* is identical with that outlined for treating the artificially fed. The supply of breast milk should be maintained and the child allowed no other food. After

the initial starvation and water feeding, it is usually necessary at the onset and in the more acute phases of the attack to limit the time of feeding and to increase the intervals between nursings. The first day or two following the fast, it is also wise to give the child water immediately preceding the nursing in order that the food may be diluted and the 24 hour ration reduced. This is also a very important procedure, in the later stages where there is much slough and pus in the evacuations and where excessive flatus gives us reason to suspect the presence of large numbers of B. Welchii in the intestine. The water feedings should be continued for 3 or 4 days, and with the return to full breast feeding, at 4 hour intervals, it is of advantage to give 2 or 3 ounces of a 5 per cent sugar solution in each interval between breast nursings.

If the child is weakly or collapsed, or if it is the subject of profound anorexia, the milk must be expressed from the breast and be fed diluted. When anorexia is extreme, recourse may be had to gavage of the expressed milk; or the child may be fed with a Breck feeder or with a medicine dropper. In order to insure a continuous supply of mother's milk, the breasts must be emptied at fixed intervals. The breast pump in the hands of most mothers is not a satisfactory means of emptying the breasts. Any woman can be taught to express the secretion of the breasts by manipulation. If this be properly done, no injury can result to the glands or nipples, and after a few trials the mother will become thoroughly expert. (See Chapter on Methods.)

The **chronic diarrheas** are much less frequent and, in most cases, less important during infancy than are the acute types. A very large proportion of them date from an antecedent attack of acute diarrhea.

They can be divided into two classes: *continuous* and *recurrent*. Rolleston and Nabarro have shown that the dysentery bacillus can become deeply lodged in the intestinal mucosa and cause recurrences in certain kinds of diarrhea which follow an earlier attack of dysentery. In attacks of recurrent diarrhea, especially when blood and pus are marked features, the patient's serum should be tested against cultures of B. dysenteriæ for its agglutinating power; if this is found to be positive, appropriate antidysentery serum should be given.

Some of the postinfectious subacute or chronic diarrheas are proteolytic in origin and have arisen through the overgrowth of this type of intestinal flora which is encouraged by

the profound changes incident to the infection. The *treatment* is essentially the same as that of the acute types of proteolytic diarrhea. However, the response to treatment is slower, and patience and skill must be exercised, for too vigorous methods of procedure may bring about the development of a food intolerance.

Once the type of diarrhea is clear, a purge of castor oil should be given. For a few days a daily bowel lavage of 2 quarts of a 5 per cent sodium bicarbonate solution is desirable; 6 or 8 ounces of this solution should be run into the bowel and siphoned out and the process repeated until the 2 quarts of solution are used. Milk, meat and egg white are rigidly excluded from the dietary. For the first day or two, a 10 per cent lactose or dextrin solution alone is used unless there is reason to suspect the presence of many B. Welchii in the stools. This suspicion should be verified in the laboratory if possible. In the presence of these organisms, starvation regime should be instituted for 24 to 48 hours, to be followed by sugar solution feedings for a like period of time.

For the next week, carbohydrate feedings of toast, cereals, gruels, strained porridges, puddings, fruit jellies and smooth fruit sauces with added lactose may be given. A little later, egg yolk and very smooth purées of vegetables, such as cooked lettuce, spinach, carrots, artichokes, peas and string beans, are added to the dietary. Still later, purées of dried peas and lentils may be used; at the end of a month it is usually safe to return to the use of egg white and meat, and after 6 weeks milk may be given. In beginning the use of milk, it is wise at first to give dilutions of top milk, pasteurized or boiled. It is the writers' practice to begin with 5 ounces of the top 10 ounces in a 20 ounce mixture to which 1 ounce of milk sugar has been added.

During the course of treatment, an occasional dose of castor oil is given, and it is of advantage to use a powder containing atropin, powdered calcium carbonate and diastase. The atropin ($\frac{1}{1200}$ grain for a 15 pound child) is given for its antispasmodic effect; the calcium carbonate (5 grains) for its effect in stimulating pancreatic and intestinal secretion and for its aid as an emulsifier of fat, and the diastase (5 grains) to augment the powers of starch digestion.

Another group of *chronic diarrheas,* which includes the variously called *"starch indigestion," "mucous disease"* and *"chronic saccharolytic diarrhea,"* is not uncommon during the second year but

is more frequent later in childhood. It is characterized by excessive flatulence, by odorless or slightly acid-smelling stools, and by the painlessness of the evacuations which contain much visible mucus. It is typical of this form of diarrhea that the bowels are immediately stimulated and the desire for evacuation becomes urgent as soon as the patient begins to eat. If the children are old enough to talk, they complain of pain about or above the umbilicus before and during evacuations; younger infants scream or give other evidence of abdominal distress.

Examination of the iodin-stained stools of these patients reveals large amounts of free unsplit starch. Many times, what appears to be mucus in quantity is merely a mass of colloidal starch. The bacteriology of the intestinal tract in this type of diarrhea has not been satisfactorily determined, but the predominating organisms are not proteolytic.

The *treatment* is essentially dietetic. Potato is an especially bad food for these patients because of its resistant starch capsules; in mild cases, the complete withdrawal of potato, banana and the coarser cereals from the diet will effect a cure. The dietary regimen depends upon the presentation of sufficient calories with little carbohydrate in the form of starch and a minimum amount of sugar. Boiled milk, meat broths, protein-milk, buttermilk and eggs form the basis of a satisfactory diet. For babies past the sixteenth month, the addition of scraped beef or mutton and cottage cheese to the dietary is of value.

A powder of atropin, calcium carbonate and diastase is also of some value. Purgation has little effect on these cases although no harm can be done by following the English practice of giving mercury and chalk in ½ to 1 grain doses 3 times a day for a week or two. John Thompson recommends Fowler's solution in 1 minim doses before meals.

Pancreatic insufficiency (celiac disease) may express itself as a severe indigestion of acute onset and protracted course characterized by the evacuation of bulky, pale gray, greasy stools, occurring fairly often in later infancy. Marked wasting and periodicity of the symptoms are features of the disease. The stools invariably contain a large amount of wasted fat which is excreted unsplit; in the graver examples of the disease, starches are often passed undigested, and sometimes proteins also escape enzyme action. It is believed that the primary infection is either a duodenitis followed by a secondary involvement of the pancreatic ducts, or that it is due to a specific change in the intestinal mucosa, interfering both with digestion and with absorption.

Recent bacteriologic studies have proved that in the intestines of many such invalids the proteolytic type of flora predominates; however, about 30 per cent of the patients conform to the findings of Herter's studies and present large numbers of aciduric organisms.

A careful investigation will reveal that most of the cases, if not all, follow an attack of acute diarrhea although there may have been an interval of several months between the onset of the chronic phase and the cessation of the acute.

The observation has been made in Karl Meyer's laboratory that in certain dogs suffering from proteolytic intoxication the pancreas is markedly fibrosed. This fact may become the starting point of the elucidation of pancreatic insufficiency.

The *treatment* is chiefly dietetic. Herter discovered that dextrins are invaluable in the dietetic management of such cases. He advised the use of thoroughly toasted breads and dextrinized crackers, powdered and cooked into a gruel.

A good many of the chronic cases have been fed upon protein-milk sometimes with benefit, but more often with a bad influence on the course of the disease—a fact which goes to prove the observation that a like clinical picture may arise from unlike etiologic conditions.

Logic demands the withdrawal of fat, especially in the type of case where the pancreatic function is definitely diminished and much free fat appears in the stools. In a mild case, the use of a thoroughly boiled skim-milk enriched by dextrins and sugar often is promptly successful. For the reason that relapses are frequent, it is essential to keep the proportion of skim-milk in the feedings low and to maintain the food fat-free for at least six weeks or two months. The withdrawal of the fat-soluble vitamines for a moderate period of time, need not alarm the medical man. They are probably not utilized in this type of case even when ingested, and the presence of fats in the intestine is damaging.

On returning to the use of fat, it is well to begin with a 33 per cent emulsion of cod liver oil, for this oil is better utilized in the intestine of a child that suffers from pancreatic insufficiency than is any other fat. At the commencement of its use, ½ a dram 4 times a day is a large enough dose for a 20 pound child. This amount may be gradually increased to 2 drams given at the same intervals.

Great care must be taken not to overfeed these children. As improvement occurs, starchy foods may be introduced; espe-

cially valuable are vegetables, well cooked and finely puréed. Following Abt's recommendation, we have used a nut-butter grinder in their preparation: the resulting pulp is a smooth, homogeneous mass, readily digestible. Very young carrots, lettuce leaves, selected spinach, green peas and artichoke hearts, where available, are the vegetables that may be used at first. As a child reaches its sixteenth month, a small amount of lean beef or mutton, very finely pulped may be added. Remembering the possibility of tape-worm infection, it is well to cook this meat rather thoroughly. This may be done on a dry pan or by dropping small portions of the pulped meat into boiling bouillon. At this age, thin bread and crackers thoroughly toasted, and zwieback may be given. Fine puréed fruits and fruit-jellies are valuable additional foods. Herter advised the use of gelatin for its caloric merits although he realized its lack of utilizable protein. It is a valuable vehicle for the administration of lactose and saccharose, and if flavored and colored, it is eagerly taken. Little by little, the child may be brought back to a rational diet; during this time it is wise to limit the intake of milk and to return to its more liberal use by gradual stages.

In the more severe cases which reach the physician, anorexia is usually extreme, the child is tremendously wasted, presents marked abdominal distention, is very irritable and sleepless and has great tenderness of its tissues. The evacuations are bulky, very pale, have a greasy appearance and reek of butyric and other fatty acids. Large amounts of mucus are seen in the stools. The physician is rarely confronted with more pitiable specimens of babyhood.

The management of severe cases demands great patience. Castor oil is not advisable for the initial purge; a mercurial or a saline evacuant is more effective. Milk of magnesia or a solution of sodium sulphate in divided doses, may be given. (One dram of the former or 1 dram of a 10 per cent solution of the latter every half hour until 12 or 15 doses have been taken). It is a good thing to add spirits of chloroform, glycerin and chloroform water to the mixture for flavoring and to prevent griping.

The use of a 2 to 6 quart bowel lavage at a temperature of 112° to 115° is good practice. Tincture of asafetida may be added to the water if there is much flatulence. This lavage should not be repeated more than once a day; if flatulence is not great, it is better to use the bowel lavage only every second day. There is rarely vomiting unless food is forced; but if this symptom should

appear, washing out the stomach with a 5 per cent sodium bicarbonate solution is indicated.

Maintenance of body-heat is an indispensable part of the treatment, for many of these babies tend to have a subnormal temperature. This fact, however, does not warrant overclothing, especially during warm weather. A daily weak mustard bath or pack followed by a simple warm pack for one hour and by a subsequent gentle body friction is also of great value and comfort to these patients. Frequent change of position is imperative if we would avoid bedsores, and for the same reason scrupulous care must be taken to prevent the bed clothing from becoming wrinkled. Every effort should be made to see that the mattresses also are of proper consistency. As in all other chronic conditions, it is important to keep the weight of the bedding from the child, especially from pressing on the toes. Much discomfort, evidenced by intractable crying, has its origin in foot cramps brought on through an oversight of this minor detail.

In the severer cases, whole milk must be absolutely excluded from the diet, and it may be months or years before such a patient will tolerate even a small amount. Likewise fats in any other form are to be interdicted. Food intolerance in such cases is sometimes absolute; and in order to coax the child above the line of tolerance, we must be content to feed him for some time on a most insufficient ration. Under these circumstances, it becomes doubly necessary to insure a proper water intake; fortunately, these patients drink water freely.

Many such a patient will utilize for his energy needs nothing but dilute sugar solutions; therefore, following the preliminary purge, it is necessary to give 5 per cent sugar, preferably anhydrous dextrose solution, during the first day or two of treatment. This is absorbed high up in the intestine and can be used in greater quantity than any other carbohydrate. Corn syrup, sometimes employed is not so useful. The use of some carbohydrate is essential if acidosis is to be escaped. The tendency toward acidosis will be combated and the palatability of the solution improved, if we use the citrates and malates in the form of fruit-juices. A solution of anhydrous dextrose may be flavored with orange, apple, raspberry, or grape juice; their slight cathartic action will be of advantage rather than otherwise.

It is imperative that the period of partial starvation should not continue too long; after 48 hours, dextrin should be added to the fat free lactic acid milk. There are infant foods made up largely of dextrin or it may be prepared by treating wheat flour

by heat in a slow oven or by thoroughly toasting white bread and reducing it to a powder and retoasting. Even with these additions, one may not be able to meet the caloric needs of the patient, and there may still be a persistent loss of weight; but under such treatment the stools should have diminished in number and have changed in consistency.

Next to be added to the diet are the starch jellies. These must be used sparingly at first and the result of their addition closely observed. As a rule, however, they are well tolerated. Arrowroot, rice flour, cornstarch, sago or tapioca jelly are useful. The latter cereals are made smooth by passing them through a fine sieve; they may be sweetened with sugar and flavored with a flavoring extract or a fruit syrup. At this time too, a fruit jelly such as currant, quince or apple may be given rather freely. One or 2 teaspoonfuls of these conserves may be given every 4 hours and may be served with the starch jellies without harm. Broths are not recommended, although they may be used as vehicles to carry the dextrins and starches; the appetites of some children are whetted by the flavor of the broth. If this dietetic regimen is well tolerated, the treatment for severe cases is the same as that outlined above for the milder cases except for the rigorous exclusion of milk.

Another most perplexing type of chronic diarrhea, characterized by *intestinal intolerance to carbohydrates*, is occasionally encountered. Children subject to this disorder show marked and persistent intolerance to carbohydrate foods in any form. Their stools contain much fat, but Howland has pointed out that in spite of this wastage, the ingestion of food fairly high in fat concentration seems to do them little harm. Such patients evacuate their fat merely because of the rapidity with which the content of the intestine is passed along. Neither fat splitting nor fat absorption is very greatly impaired.

Dietetic *treatment* here resolves itself into the withdrawal of carbohydrates in accord with the principles already laid down for the treatment of acute saccharolytic diarrheas. In diarrheas of this character, the proper use of protein-milk is followed by positive and gratifying amelioration. The return to a normal diet must be made gradually through some months. In the meantime, a high but diminishing protein ration should be maintained. Broths and meat pulp may be utilized; after many weeks as the symptoms abate the starch jellies are added, and later a small amount of well cooked, smooth cereal may be used. These additions may be followed by the use of smooth vegetable pulps, and finally the child may be returned to a normal diet.

CHAPTER IV

CONSTIPATION

Constipation in young infants is frequent and annoying though it is rarely a matter of serious moment. It occurs in the breast-fed as well as in the artificially fed. For purposes of treatment, the condition may be considered under three heads: First, that in which the action of the *upper bowel* is involved; second, that in which the expelling apparatus of the *lower bowel* and rectum is at fault; and third, that in which the constipation is a result of some *constitutional disturbance,* especially of the muscular or nervous systems.

The young infant is born with a nervous system imperfectly integrated and time is a factor in the development of integration. Therefore the normal nervous impulses essential to the proper physical action of the intestine may not develop for several months. This failure may affect the upper or lower bowel, most often the latter. The type of constipation in which soft stool fills the rectum, but is expelled only after manipulation or irritation of the anal canal, is evidence of the failure of nervous co-ordination.

This failure may lead to a second source of constipation. The work of Hertz has accentuated the rôle played by the reverse peristaltic wave in the large intestine. This wave mixes the contents of the transverse and ascending colon and returns the liquid fecal accumulations to the cecum where its fluid portion is readily absorbed. There is an optimum of absorption advantageous for evacuation. However, when too much fluid is abstracted and the contents become hard and dry, there will be great difficulty in ridding the bowel of these masses. In the adult, such masses are apt to be evacuated with a fair degree of rapidity, but in a baby the lack of nervous control often results in spastic contractions of the gut and a further aggravation of the constipation. Furthermore, the anatomic peculiarity of the sigmoid or as Treves descriptively calls it the "omega loop", overlong in proportion to the rest of the intestine and ill supported, adds another element favoring retention of feces in the large intestine of infants.

The character of the feces themselves is a further influence

favoring constipation. This is especially true for infants that are artificially fed. It has long been recognized that stools containing a high proportion of soaps are difficult of evacuation. Protein-milk feeding depends in part for its success on the production of just such stools. The normal peristaltic activities of the bowel are in abeyance when these calcium soap stools are present, because they inhibit the growth of aciduric bacteria, which if they occur in proper proportion, produce a constant but mild flow of acids that stimulate the intestinal movements and aid evacuation. Such soap stools occur in the presence of a food combination, high in both protein and fat. The limitation of one or the other of these with an increase in some carbohydrate pabulum often will be sufficient to overcome the constipation.

In the *treatment* of this type of the constipation of artificially fed infants a modification of Kellar's malt-soup formula is almost always effective; the modification consists in preparing a formula containing an amount of milk proportioned to the child's nutritional needs. To this is added the malt-soup extract and flour in quantities needed to provide a properly balanced ration. Kellar's original formula and the formulas printed by the malt-soup extract manufacturers are undesirable because they bear no relation to the nutritional needs of the individual infant. The method of preparation of malt-soup is given in detail in the chapter on Formulas.

For the milder types of constipation, the malt-soup extract without being made up, may be added directly to the feedings ½ dram to each of 2 or 3 bottles daily, according to the needs of the child. It is difficult to manipulate the malt-soup extract but an easy way to manage its addition is to put the required dose into the bottom of a small wide-mouthed pitcher and have the contents of the bottle warmed and poured out into the pitcher. The mixture may then be stirred until the malt-soup extract is dissolved and it is then returned to the nursing bottle and given to the baby.

The same sort of constipation is one of the most frequent troubles of the breast-fed. For these babies too, malt-soup extract is often an excellent remedy. One-half teaspoonful dissolved in ½ ounce of water and flavored with a few drops of orange juice, is to be given before nursings. Usually, if it be given 2 or 3 times a day, the constipation will be corrected. The greatest objection to the thick malt-soup extract is that a proportion of infants, perhaps 10 per cent of them, will vomit

any mixture containing it. In such cases, some other remedy must be employed. A glass rod, the soap stick or a gluten suppository is easily used and harmless although a bowel habit may be established which is difficult to eradicate. Glycerin suppositories are not advised because if persistently used for any length of time, they may set up a proctitis. The same objection holds for glycerin enemas. For those patients in whom hard fecal masses larger than can be evacuated with comfort, pass into the rectum, there is nothing so effective as the injection and retention of one or two ounces of paraffine oil or cotton seed oil, night and morning. If given slowly and it is retained, the fecal masses are softened and then evacuated with ease. In this way, tears of the mucous membrane may be prevented. Tears and ulcerated anal fissures provide one of the commonest sources of pain, producing intractable crying in infants.

For those children who cannot take the malt-soup extracts. other forms of carbohydrates may be used, and the proportion of either fat or protein diminished in the feeding mixture. Some prefer to keep the proportions down by using top-milks in high dilution and making up the strength of the food by the use of cereals. Of these, rye flour is the most useful for a baby that is constipated. The top-milks are to be used so that the fat content of the mixture will fall between 2 per cent and 3 per cent. In the period before such feeding has become effective, the constipation may be relieved by giving ½ to 1 teaspoonful of one of the heavy petroleum oils 3 or 4 times a day after feedings. This preparation is invariably well taken with or without orange juice.

Too much stress cannot be laid on the need for instruction of the mother that castor oil as commonly used, is productive of constipation and that infants do not need routine doses of laxatives. If more mothers were taught that a coated tongue and foul breath usually are the result of infections about the pharynx and nasopharynx, or uncleanliness about the mouth or nose, the laxative habit could be minimized.

Castor oil, one of the most valuable drugs in the pharmacopeia, should be retained by the physician for combating diarrheas according to his judgment. The same should be said of the mercurial purges. Milk of magnesia though unquestionably effective in producing evacuations in this or in any other type of remediable constipation of infants, is an agent produc-

tive of colic; abdominal pain often follows its use, especially when insufficient dosages are given. Neither milk of magnesia nor any other laxative should be recommended to mothers for general use. Only rarely will a properly devised diet fail to remedy a constipation that is amenable to relief by milk of magnesia.

While there is no more useful procedure in the treatment of infants than the use of enema, there is nothing more pernicious than the enema habit. Glycerin and soap suds enemas are frequently the causes of proctitis. The hard points of the syringes may injure the mucous membrane and set up ulcerations and fissures. Both of these conditions may contribute to the retention of feces in the sigmoid. The continued use of irritating enemas may so affect the nerve-end distributions in the bowel that they fail to respond without extraordinary stimulation, and so the habit of constipation is set up. On the other hand, when a child is already seriously constipated and the rectum is filled with feces, to the discomfort of the baby, a low enema of glycerin and water, oil or soap suds will cause an evacuation and will relieve distress and quiet the crying. The high enema should be retained for its value in bowel flushing, and should never be used for the relief of constipation. Its proper office is as a therapeutic measure in diarrhea. Used at a temperature of 112° or 115° with the addition of bicarbonate of soda and an antispasmodic such as asafetida, it is of value in the relief of abdominal distress, but the need for its repeated use will rarely occur and habit cannot be formed by the use of an occasional colonic lavage.

Many children who have difficulty in passing a normal or slightly formed stool, while they are lying in bed will easily evacuate the bowels if they are held in an upright position. This is one reason why a child ought early to be trained to have a daily evacuation into a vessel. Many babies can be trained to clean diapers as early as the fifth month. The vessel is placed between the attendant's knees, the child well supported in the erect position with its back against the nurse's body, the baby's thighs are bent against the abdomen and held in that position by a firm and gentle pressure. During the first few attempts, soap stick, glass rod or gluten suppositories may be inserted. In a few days the child will learn the meaning of the unusual position, the passage of the stool will be accomplished and the habit of daily evacuation established. The establishment of such a habit is one of the most effective means of preventing constipation in later infancy.

In the second year of babyhood, when the child is able to sit on a toilet chair or vessel, care should be taken that mechanical advantage for defecation is not lost by bad position. It must always be remembered that the levator ani as an agent of expulsion must have a firm fulcrum in a well supported ischial tuberosity. A child sitting on a vessel or a toilet chair that is too large, loses this mechanical advantage and finds difficulty in emptying the anal canal. The same disability follows the use of nursery chairs that are too low or toilets that do not allow the child's feet to reach the floor. The child at stool should sit well supported on a seat with an opening neither too large nor too small, and in such a position that the thighs are flexed but not to excess and if possible with the feet on the floor. It should be possible for the child to lean forward so that the body can be brought to an acute angle without a sense of overbalance. In a surprising number of instances of constipation that occur during the second year, minute attention to the details of mechanical advantage will be rewarded by a cure.

Massage as a means of relief of constipation is a valuable adjunct but alone it seldom effects a cure. It is useful in stimulating the intestine and it favors the passage of the intestinal contents particularly from the colon. It should be gentle and applied in such a manner that the manipulation follows the anatomical outlines of the large intestine.

Most cases of constipation that occur during the first year of life can be traced to faulty *lower bowel* mechanism or improper feeding. Toward the end of the first year and during the second year, the defective regulation of the upper bowel becomes a frequent cause of constipation. While there are a few cases in which the lack of tone is a physical fault, in a great majority of instances as in adults, overtone and ill-regulated nervous impulses set up a spastic contraction and so prevent the proper passage of fecal matter into the lower bowel and favor its retention until the absorption of the fluid portion is excessive. The presence of overdry or hard scybalæ in turn calls forth further spastic contractions and aggravates the constipation. A properly balanced diet, especially devised to keep fat or protein low, given with a maximum of carbohydrate is a valuable dietetic measure, but alone it is rarely sufficient to relieve constipation. The attempt is often made to overcome this type of constipation by the use of irritating foods such as bran and coarsely ground grains which carry a large amount of insoluble residue and by the ad-

dition of such things as flaxseed and charcoal. These additions to the diet may be effective for a time, but sooner or later the irritation they produce will increase the spasticity and aggravate the constipation. Granulated agar or agar-jelly, used for its hygroscopic effect is not open to these objections. A good grade of New Orleans molasses or of sorghum is often an effective addition to the diet. A teaspoonful of either added to each 2 or 3 bottles of a nursing baby or a dessertspoonful or two added to the cereal of an older infant, often succeeds in relaxing the spastic bowel. For the correction of constipation in the breast-fed, molasses or malt extract may be diluted with water and be given before nursing.

It may be necessary when dietetic measures fail in spastic types of constipation to have recourse to drugs. In such instances atropin in doses of $\frac{1}{2400}$ of a grain for a 15 pound baby, is ready to hand as a most useful antispasmodic. It may be given in watery solution alone or in combination with powdered calcium carbonate or calcium lactate. (Calcium stimulates intestinal secretion and encourages fat emulsification). If there is much gas and distention present, animal charcoal is a valuable addition to the mixture, and when anemia also is striking as it often is, the addition to this powder of a few grains of the saccharated carbonate of iron is in order. Cascara has been very widely and effectively used but the continued administration of this drug or any other cathartic is to be deprecated. Too often, the cathartic prescribed for a child is continued over a period of months or years so that it becomes impossible to retrain the bowel to normal function. An effective dose of cascara is 10 drops of the fluid extract for a 15 pound baby. When it is necessary to give cathartics to children, it is better to give small doses at more frequent intervals than to give massive doses at one. Ten to 15 drops of aromatic cascara given in cold water before feedings will be more effective than 1 or 2 drams given once daily. Little by little, the dose can be diminished and ultimately, one by one, the doses withdrawn so that in this way the child may be trained to have normal evacuations. The common practice of giving children "graham crackers" should be discouraged.

The idea that a cure of constipation in the breast-fed can be achieved by giving the mother drastic purgatives is to be condemned. The amount of the drug excreted through the mother's milk is variable and there is no way of controlling the dose that the child gets.

Hernias, tuberculous peritonitis and *appendicitis,* any or all may interfere with normal peristalsis and so produce constipation. In the condition known as *megacolon,* (Hirschsprung's Disease), in which the colon is greatly dilated, constipation is a part of the clinical picture. On the other hand, it is improbable that a dilated colon ever results from constipation or fecal impaction.

Prolapse of the rectum may occur as the result of straining or from pressure exerted by fecal masses as they descend into the rectum. However, it can only happen in the case of a child who has some anatomic peculiarity, such as a redundant sigmoid or an overlong rectum. These children are invariably thin and wasted and without the normal support produced by the adipose tissue in the rectal and perirectal spaces.

The *treatment* of the prolapse is simply the treatment of constipation and the correction of spasticity. The provision of a diet which will fatten the child and at the same time produce a soft fecal mass for evacuation is essential. The prolapse is less liable to occur if the child is lying flat on the back during evacuation. Such a child, if old enough, should be taught the use of the bed-pan. Some advantage may accrue from the strapping of the buttocks with a broad band of adhesive plaster long enough to pass from one anterior superior spine across the buttocks to the other. It is important that the prolapsed intestine be returned immediately on its protrusion. The attendant should watch the act of defecation and as soon as the gut appears it should be replaced. Under no circumstances should the child be allowed to strain while the viscus is protruded. The rectum is easily returned by grasping the mass with the five fingers and gently squeezing it before the attempt is made to push it back into the pelvic space. One or two minutes of gentle pressure will force the blood out sufficiently to reduce the size of the mass so that it may be returned without force. At the beginning of treatment, it may be necessary to return the bowel as often as 4 or 5 times during one act of defecation. However, if these directions are followed carefully, after a few days extrusion will be infrequent and will gradually cease to occur.

Mental defectives, especially cretins, suffer from constipation. In the group in which cretinism is the basis of deficiency, thyroid treatment is remarkably effective in relieving constipation. It is often impossible to induce mental defectives to take proper diet, so that the use of drugs becomes necessary. However, every

attempt should be made to control the bowel function by dietetic means.

In many nutritional disturbances, constipation is a troublesome symptom. In *rickets,* this is especially true. On the establishment of a proper regimen with the improvement of the bowel muscle tone, the constipation improves most often without the need for any cathartic drug treatment. Massage and passive movements of the thighs are invaluable aids in restoring the muscular tone. Cod liver oil emulsion 1 dram 3 times a day and phosphorus ($\frac{1}{400}$ grain 3 times a day for a 15 pound baby) are useful in this condition. The cod liver oil itself has an excellent effect on the intestinal contents, rendering them softer and more easily evacuated.

Anemia is a frequent complication in constipation of rachitic children and even in those without rickets, this symptom is often evident. In such cases iron and arsenic are strikingly remedial. One of the most successful means of giving the combination is as cacodylate of iron, by intramuscular injection. The sterile ampoules, commercially available, provide a safe and effective means of medication. Five to ten minims doses given daily for 6 or 7 days, then every second day for a period of 10 days may be administered. If improvement is not evident, a second similar course may be undertaken after a rest of 2 weeks.

Bottle-fed babies get a great deal of fluid with their formulas and certain waters, because of their high content of alkali earths known as "hard," become undoubted factors in the production of constipation. The quality of the water should always be observed. Distilled, or soft water should be used if necessary.

Constipation is a symptom in cases of *bowel obstruction* and its treatment in such a circumstance lies in the relief of the obstruction.

Summary

Confronted by a persistent case of constipation in a breast-fed infant we should:

Make no attempt to combat the disorder through drugs given to the mother.

Avoid giving drugs to the infant if possible.

Attempt dietetic control by supplementary feedings of malt-soup extract in water or diluted orange juice (one-half dram to the ounce before one or more nursings daily).

Before beginning the dietetic treatment, give a single sufficient dose of milk of magnesia (1 to 2 drams to a 15 pound baby).

Replace one feeding a day when the child is over 5 months, with a feeding of rye flour gruel or cream of rye gruel, sweetened with a little molasses and enriched with butter.

Instruct the mother in the methods of abdominal massage and of obtaining proper posture for defecation.

Provide the mother or nurse with a rubber finger cot and teach her gently to dilate the sphincter with a well lubricated little finger or to use a gluten suppository or a bent glass rod.

These things failing, we may be obliged to resort to the use of drugs and give heavy petroleum oil in doses of ½ to 1 dram before two or three of the nursings a day. If it is absolutely necessary to resort to the use of the purgatives, milk of magnesia in doses of 1 to 2 drams added to sweetened water or orange juice may be given. Cascara may be used; or if the stools are very hard and dry tincture of podophyllum. In those cases in which it is found essential to give drugs, a persistent attempt should be made to withdraw the laxatives and purges by progressively diminishing doses over a period of weeks. With a few modifications, this plan may be followed in the treatment of constipation in the bottle-fed. For constipated infants, we may add the malt-soup extract directly to the feedings; the gruel may replace a bottle feeding instead of a nursing; and the milk of magnesia used may be added to the bottle. The use of podophyllum will be more frequently indicated because the constipated stools of the bottle-fed are more often dry and friable. In addition it often will be necessary at the onset of a course of treatment to withdraw milk entirely for one or two days and to give the baby only decoctions of carbohydrates.

When the infant is older, posture while defecating and regularity of habit are more readily controlled. A sufficient water and food intake should be assured, and the early establishment of a mixed diet is indicated. The vegetable and fruit pulps are to be given freely and it is necessary that sufficient carbohydrate be provided to establish a preponderance of aciduric organisms in the intestine. At this age, the use of enemas and suppositories is pernicious. If the use of drugs is unavoidable, the aromatic extract of cascara with nux vomica in the atonic types of constipation and of atropin in the spastic, are the most valuable remedies. Either should be given in small doses before several feedings rather than in one daily dose and each should be gradually withdrawn as the bowel becomes trained to function without assistance.

CHAPTER V

NUTRITION

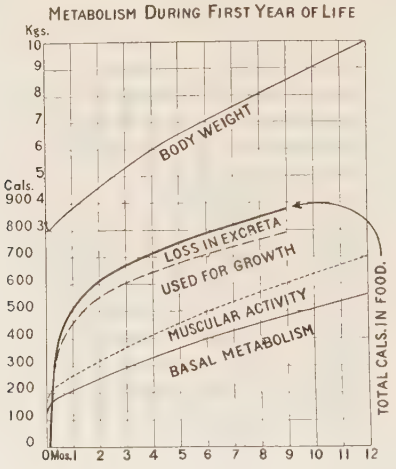
No subject has been made more complex than the study of infant nutrition. This complexity has followed from the fact that thought and writing on the subject rested on no firm physiological foundation. During the last ten years, little by little, facts have been brought to light, each producing a modification of previously held ideas but none sufficiently decisive at once to bring a readjustment of our views. As in every other field where investigation has proved fruitful, much controversy followed the presentation of new ideas and we were confronted with "percentage schools of feeding," "caloric schools of feeding," "Boston methods," "Finkelstein methods," and many others, each school contending vigorously for its own point of view, and each contributing essential knowledge to the subject. Today, while much remains to be elucidated, thanks to the different groups of men who have given their energies to the subject, it is possible to formulate a simple, uncomplicated practice from the knowledge in hand.

Little need be said in a book on the management of the sick infant, about the nutrition of the normal child further than to state the facts necessary for a reasonable consideration of abnormal nutrition. A clear conception of the fundamental metabolic needs of a child can be best obtained by studying Fritz Talbot's metabolic charts. These show the relation of total calories ingested to body weight and to basal metabolism, and furthermore they reveal in a graphic way, the proportions of food that are lost in excreta and through muscular activity, and the proportion that is used for the purposes of growth.

These charts show that with a wide range of variations for the individual, a baby needs on an average, 500 calories a day in food by the end of the first month and that this need increases proportionately with its increase in weight, running up nearly to 900 calories at the end of the ninth month. Of the energy intake, 90 per cent is available for the purposes of the body and 10 per cent only is lost as excreta. Muscular activity, kicking, crying and restlessness of all sorts, may use up as much as 25 per cent of the available energy supplied by the food; basal

metabolism requires about 40 per cent; so that only about 25 per cent of the energy of the ingested food is utilized for purposes of growth.

Such are the facts upon which we estimate the amount of food needed for a normal baby. Recent investigations have shown us that we must take into consideration factors other than the mere energy needs of a child. The work of McCollum, Alfred Hess and Osborne and Mendell show clearly that a consideration of energy needs alone is not a sufficient guide for the preparation of the food for infants. We must consider the child's needs for certain unidentified chemical substances which



The caloric requirements of normal infants and children from birth to puberty. (Fritz B. Talbot, American Journal Diseases of Children, Vol. 18, No. 4.)

occur in fat, in the leaves of green vegetables, in tomatoes and in citrus fruits. At the present stage of our knowledge, it would seem that these substances play some undetermined rôle in stimulating normal metabolism and in promoting proper growth. While these substances (*vitamines*) are most important in infant feeding, there is little chance that infants will be deprived of them, for they occur abundantly in milk fat, and egg yolk, and in spinach and other green leaf vegetables which are commonly used in infant dietaries. One of the objections to the exclusive use of proprietary foods is their lack of these "vitamines," although this objection cannot be justly raised against those of the proprietaries which call for dairy milk in the preparation of formulas.

In order to supply the energy needs of a normal child, it is

necessary to keep in mind the caloric values of the various foods suitable for use in feeding infants.

TABLE OF APPROXIMATE CALORIC VALUES PER OUNCE OF FOODS

Whole milk	20
1. Upper 24 ounces cow's milk	22.5
2. " 16 " " "	25
3. " 10 " " "	35
4. " 5 " " "	55
Skimmed-milk	12
Butter-milk	12
Protein-milk	12
Condensed-milk, sweetened	110
Condensed-milk, unsweetened	55
Milk-sugar	135
Cane-sugar	135
Dextro-maltose	135
Nestle's Food	115
Corn syrup	120
Malted milk	135
Imperial Granum	120
Eskay's Food	128
Mellin's Food	120
Barley flour	120
Wheat flour	120
Rice flour	120
Oatmeal	135

1, 2, 3 and 4 dipped from a quart bottle of milk that has stood on ice from 6 to 8 hours.

A study of the caloric values of the foods alone will not suffice, for the digestibility and availability of the different forms of foods must be taken into consideration and be allowed for. Although the reason is not generally understood, it is a very widely accepted fact that most full weight normal infants do not do well if they are given more than 2 ounces per pound per day of cow's milk. It is also clear that high concentrations of carbohydrates and of fats are badly tolerated and that the tolerance of each child for the different food-stuffs is a personal matter which varies from time to time without known reason. Julius H. Hess has devised a very simple and effective scheme for feeding normal infants; after due consideration of the metabolic requirements of infants and the caloric values of available foods, he has worked out a relationship between the body weight and the quantities of different food elements required. He has come to the conclusion that a safe minimum for a healthy infant is approximately 1½ ounces of milk per pound per day; that this amount of milk presents to the baby about the amount and proportion of fat and protein best suited to the average normal child; and that the amount of carbohydrate to be added should

be for most children, $\frac{1}{10}$ of an ounce per pound of body weight per day. Hess uses cane sugar by preference. When other sugars, maltose or dextrins are used, he advises giving $\frac{1}{8}$ ounce per pound per day. In place of water, cereal decoctions may be used to dilute the milk. They can usually be added with advantage by the end of the eighth week. These are especially valuable in modifying the sweetness of the cane sugar.

Certain babies deviate from the usual in their reactions to their meals. Some are unable or unwilling to accept the amount or concentration of food that practice has proved most applicable to most children of the same age and weight. Others, while accepting the feeding mixtures, react in an abnormal manner. Properly balanced foods given in proper quantities may be followed by no gain or even by a loss in weight, and at the same time digestive disturbances such as vomiting, diarrhea, flatulence and colic, may occur. This phenomenon has been designated by Finkelstein and his followers as a "paradoxical reaction," and it is thought by them to be due to some unexplained events that happen during "intermediary metabolism." It is a more reasonable opinion that this behavior on the part of the body is due to disturbances in water balance and the restitution of fluid, in most instances, will be followed by a return to normal powers of food utilization. Fluid may be given by mouth in mild cases, or by intraperitoneal or subcutaneous injections in the graver cases. The results of the therapeutic use of water will be augmented if we exclude the more complex foods for a few meals and give the child only sugar solutions (7 per cent to 10 per cent glucose or lactose). After 48 hours, a return should be made to the use of protein and fat by adding small quantities of milk to the sugar solution. Day by day the amount of milk used is to be increased until by the end of a week the child is receiving a properly balanced formula.

No satisfactory classification of nutritional disorders on an etiologic basis is possible at the present time because many of the fundamental factors in the production of these disturbances are unexplained. And yet, the classification of Finkelstein which avoids etiological considerations as a basis, is hardly more satisfactory, because it groups many entirely unrelated conditions together under a single head for discussion. The terminology used conflicts with the more general use of similar terms in America.

It seems simpler to consider these nutritional disturbances as due to:

A. Overfeeding: (too great quantity or too great concentration of individual food elements).

B. Underfeeding.

C. Variations in the infant's digestive power.

D. Variations in the infant's metabolism.

E. Bacterial processes in the intestine, fermentative or proteolytic.

Overfeeding.—There are three tests that will tell us if the child is receiving too much food—its discomfort, the character of the excreta and an estimation of the number of calories ingested. In this connection, it is important to consider the food that should be available for nutrition. The percentage method of feeding is open to the criticism that it does not focus attention on the 24-hour ration. However, this is a weakness not inherent in the method itself but in the mode of its application. Remembering the individual peculiarities of children, some of whom need more and some less calories per pound, for proper nutrition, it is clear that a thin child should be given proportionately more food than a fatter child, and that a restless, crying infant who is using up a large part of its food in the production of muscular activity, should be fed more than a quiet one. It is a simple matter to calculate whether a child is obtaining food of a caloric value appropriate to its weight and condition.

When the quantity of food given is a source of distress, the child is restless, cries vigorously a short time after taking his bottle and will fall asleep, perhaps one or two hours later. It is a characteristic of these children that they sleep well at night when the intervals between meals are longer and they wake up happy in the morning and that they are subject to recurring periods of discomfort which follow meals and which culminate in a crisis of crying and pain late in the afternoons.

Depending on the character of the overfeeding, these children usually evacuate numerous stools which vary in character with the stage of the disturbance and with the composition of the food. In the beginning, the stools are normal in color and in consistency and they are 3 or 4 a day in number. Later, they may become green, sometimes watery, sometimes pulpy. These characteristics depend on the reaction between the food elements and the intestinal bacteria.

Flatulence and what is called "colic" are common symptoms but true spasmodic colic is rare. Sometimes when the quantity given at each meal is too great, vomiting results. This may vary from a slight regurgitation to a projectile emesis closely resembling the vomiting of a pyloric obstruction.

All the discomforts that follow the feeding of too bulky meals may be produced in a perfectly normal child whose abdomen is swathed in a tight, inelastic band that compresses the stomach and prevents the normal distention of the abdomen after a meal. There is no logical use for a binder after the umbilical cord separates.

Overfeeding of individual elements in the food may produce similar symptoms. A mixture otherwise adequate but too rich in fat will often produce the symptoms of quantitative overfeeding, especially the late afternoon distress. Fat leaves the stomach slowly and there may be an accumulation of fatty acids which reaches its maximum late in the afternoon; this causes gastric pain, which the child resents by prolonged and vigorous screaming, by drawing its legs up and rolling its head wildly and by chewing eagerly at its fingers. Frequently vomiting is added to the picture and in these circumstances as soon as the stomach is emptied of the fatty acids and mucus which have accumulated, the child seems better. The constant sucking of the fingers and the fact that the ingestion of food temporarily relieves the distress, leads the family to interpret the clinical picture as due to hunger with the result that the child is given more food and the symptoms are thus aggravated. Hungry children never behave in this way. They are less insistent and not so apparently ravenous.

When the overfeeding is with carbohydrates, the child rarely exhibits such extreme distress as when fat is the offending food factor. The discomfort produced by sugars comes on very rapidly after the ingestion of food. Instead of frank vomiting there is rather, a continuous regurgitation of small amounts of sour smelling, watery, curdled fluid which may be acid enough to redden the mucous membrane of the mouth and lips. The afternoon crises of screaming and pseudohunger are not so apt to be a part of the clinical evidence as when fat is overfed. The crying attacks are more frequent, of shorter duration and less persistent but they are more likely to recur with frequency night and day, and so to disturb the child's rest. The stools are prone

to be many, watery and acid. This is a condition which soon is followed by disturbances of metabolism.

When the flours or cereals are the carbohydrates that are being overused, provided milk is given at the same time, the symptoms vary but little from those produced by the sugars except that the stools are at first browner in color; later they change and become characteristically acid, excoriating, and mucous containing. These qualities show that an excessive fermentative reaction has been set up in the intestine.

Overfeeding with protein is a rare occurrence in infancy. The incidence of the symptoms of such an overfeeding does not often fall upon the digestion but is exhibited by disturbances of metabolism and of the balance of the intestinal flora. The vomiting of gross curds and the appearance of large solid protein masses in the stools is not evidence of the excessive ingestion of protein but it shows that the protein of the milk was chemically in such a state that it formed large curds with the ferments of the stomach, a condition readily overcome by boiling the milk. The same end can be accomplished by alkalinizing milk with sodium citrate, or sodium bicarbonate, 1 gr. to the ounce, or by precipitating it with renin, dry or in solution.

The same evidence that shows a bottle-fed baby to be overfed, is present in the case of a breast-fed infant who is getting too much food. Overfeeding is not very common except when mothers attempt to follow a routine with strict precision and wake a vigorous, hard nursing baby at short intervals in order to induce him to feed in spite of his own better judgment. A healthy baby whose mother has an ample free-flowing supply of milk, let alone, will nurse usually 5 times a day and wake himself at intervals of 4 hours in the day and be content with 1 feeding at night during the first 5 months of his life. After that, he will often be entirely satisfied with 4 breast feedings in a day and if this schedule be adhered to, the baby will show no indication of overfeeding but will develop in the happiest way.

The *treatment* of a breast-fed baby that is being overfed, consists in increasing the nursing intervals, shortening the time of each nursing or diluting the food in the infant's stomach. The latter can be accomplished readily by giving the child an ounce or two of water from a bottle, spoon or medicine dropper, immediately before it takes the breast.

Underfeeding.—A few years ago overfeeding was a very common practice but the insistent preachments of the medical pro-

fession and the innumerable leaflets that have been given to the public have so emphasized the danger of this procedure that today, underfeeding is more prevalent. The average practical nurse and the well read mother are so fearful of overfeeding, that the babies in their charge often receive insufficient food. Such babies appear comparatively healthy but they are underweight and have a pathetic look about their eyes. They are usually very quiet. Some of them cry in a half-hearted way when their bottles are taken away, or a little while before the regular feeding time, but as infants are creatures of habit, they soon become resigned to an insufficient diet and apart from their mild emaciation and listlessness, constipation and diminished urine, they show few clinical signs.

When the infant is breast-fed and when the milk supply at first becomes inadequate, the baby will drop the breast with an angry cry or a whimper, grasp it again and finally settle down to nursing in an attempt to get all that he can of the insufficient secretion; and he will remain nursing sometimes for 25 or 30 minutes, and will cry when taken away from the breast. When the baby does not take his feeding well, and nurses continuously for 30 or 35 minutes without satisfaction, it is advisable to weigh the child before and after nursing so as to find out exactly how much milk he does get. The quality of milk is rarely a factor in determining insufficient feeding of a nursling. The withdrawal and analysis of small samples of milk or even of large quantities at one or two nursings gives no information at all comparable in value to the information we derive from observing the physical progress of the baby. This progress should be verified by weighing the amount of milk he derives from the breast at a nursing. The weighing should be repeated several times daily for a few days. Anything less than 5 ounces gain a week in the child's weight is unsatisfactory and if the stools are not uniform in number, character, color and odor, the baby should be kept under close observation. But it must not be forgotten that an infant who is doing perfectly well and who needs no treatment, may have stools that contain an excess of mucous and bile pigments or that may lack homogeniety and show white masses of soft curds. When such babies gain in weight and well-being, the mothers should be reassured but, none-theless, the attending physician should be more watchful of such infants than of those whose evacuations are usual in appearance.

When it is apparent that the mother's milk supply is giving out,

every attempt should be made to stimulate its production. Many drugs have been recommended for this purpose, but from experimental studies, it appears that there is no such thing as a medicinal galactogogue. Milk production can be stimulated in animals for short periods by the injection of pituitrin but this is compensated for by a like period of diminished supply and the amount in daily aggregate is in no way increased. General hygienic measures which include a good mixed diet, sufficient fluid, moderate exercise with avoidance of fatigue, equanimity of mind and above all sufficient sleep, are the best of *galactogogues.* Care must be taken to insure the withdrawal of all residual milk by manual expression after the child has finished its meal. This is especially important when the babies are premature or weakly and are unable themselves completely to empty the breast. The residual milk expressed may be fed to the weakly child by pipette or bottle, even if the secretion of the breast is insufficient for the needs of the baby, and complementary feedings of cow's milk have to be given; still, it is essential completely to empty the breast by manual expression. By this procedure, we are able to maintain, often to increase, the secretion of the mammæ. On the other hand, there is no more efficient way of stopping milk secretion than by allowing a vigorous baby to nurse frequently on an empty breast. The use of the breast pump is deprecated and manual expression of the breast milk urged, as the breast pump may damage the nipples, and its use is inefficient and annoying to the mother. (For technic of manual expression, see chapter on Methods.)

One, two or even three of the breast feedings may be omitted and a rational milk formula substituted. This is much better than the practice of taking the infant away from the breast entirely, as the mother's milk supply may return under this treatment; and even if it is not completely recovered, the advantage of a few breast feedings a day is too great to be forfeited, particularly if the infant is not robust. If under this treatment, the infant continues to lose weight, as rarely occurs, it is good practice to substitute all its feedings with a well devised formula of cow's milk.

The reason often given that "the milk is not rich enough" as an excuse for taking the baby off the breast, is seldom valid. The real reason for the loss in weight usually is an insufficient amount, and the practice of supplementing the breast feedings with a cream mixture in order to enrich them can only be condemned.

Variations of Digestive Power.—Each child born has an individual potential of development and parts of this potential of development are the powers of digestion and assimilation. The premature or the full term child with a *congenital inferiority,* will have less ability to utilize food in adequate amounts than the child who is born vigorous and fully developed. The weaklings at birth are those who readily drift into a nutritional state in which food intolerance is a feature, increasing the complexity of the physician's problem. It is also a well-recognized clinical fact that the *parenteral infections* reduce the infant's tolerance for foods. It is important to give the sick child a ration that it can digest. It is quite as important to watch the nutritional behavior of a child sick with an infection as it is to deal with the infection itself. Attempts to force feeding, especially by the use of fats and proteins, may result in persistent indigestion or in a grave metabolic intolerance. The same attention to nutrition is necessary in cases of *circulatory weakness* which accompany congenital affections of the heart and in those which follow such infections as pneumonia, furunculosis and otitis.

Food intolerance may be purely digestive, vomiting, diarrhea and discomfort following ingestion of appropriate amounts of apparently proper foods. If this state of affairs can develop when the child is on the breast, how much more it may be expected when we have to feed such infants artificially. Cow's milk is a mixture of food elements physiologically appropriate for the calf, a rapidly growing animal whose muscular activities are in evidence almost from birth and whose digestive apparatus is different in arrangement and much less sensitive than that of the human infant. Intolerance to human milk is a biological accident, but tolerance in the human being to cow's milk is equally accidental. Marriott has pointed out the fact that cow's milk has the power to unite with either alkalies, or acids in large amounts and to produce little change in the reaction of the resulting mixtures, while the analogous powers of human breast milk are much weaker. As a result of this "buffer" value of cow's milk, it follows that the infant stomach must secrete three times as much hydrochloric acid in order to attain the condition optimum for digestion of cow's milk as would have been necessary had the ingested food been human milk. The power to accomplish this extra secretion is not given all infants, and as renin production, bacterial inhibition, and pancreatic stimulation through the activity of acid aroused duodenal secretion, all depend upon the presence in

the digestive tract of chyme of an optimum reactivity, these facts seem to carry us further toward an understanding of why certain babies are intolerant of cow's milk. Because of the availability and relative cheapness of cow's milk, it is fortunate that the great majority of the human young are able to tolerate this food. The facts are that possibly 10 per cent of well infants, and all sick infants, acquire a toleration for bovine milk with some difficulty; that 2 per cent or 3 per cent of them acquire such toleration with great difficulty; and a very few are unable at all to accept this food stuff. It is this 15 per cent of babies that provide feeding problems for the practitioner. The lower the child's tolerance, the greater is the doctor's problem.

The chemical and physical differences between the fats of human and cow's milk which extend to the kind and quality of fatty acids and to the melting point of the fat, render the digestion of the fat of cow's milk somewhat difficult for all babies and impossible for some. *Lowered digestive capacity for fat* is made evident in different ways by different children. The irritation of accumulated fatty acids may cause emesis which has a characteristic butyric acid odor like rancid butter. If the high fat feeding is persisted in after the appearance of the warning emesis, the pyloric reflex may come into play and *pylorospasm* evidenced by projectile vomiting, pain and a mild epigastric wave may become evident. This condition sometimes is suggestive of hypertrophic pyloric obstruction, which however, may be excluded by the fact that after a few stomach washings and the provision of a food low in fat, pylorospasm will disappear. Very often, *discomfort without vomiting* or with occasional eructation is the only sign of lowered fat tolerance and the great distress of the child makes one believe that a sensory condition akin to that which adults suffer in heart burn or gastralgia, is present. Such distress when it is due to fat intolerance is always amenable to stomach washing with a dilute alkaline solution. This procedure should be followed by feedings of low fat content.

Diarrhea caused by fat is not so common as is generally thought. Any stool that has been hurried through the intestinal tract shows large proportions of unsplit fat and fatty acids. The so-called "scrambled egg" stool, it is true, contains large quantities of fat, but it is almost always an index of overfeeding with sugar. Interferences with the secretion of pancreatic lipase and of the biliary acids permit the appearance of numerous fatty

stools. This lowered digestive level for fats is not necessarily an indication of the intolerance of the body for fats themselves. It is often a temporary affair, although it may be the first evidence of a pancreatic insufficiency of the gravest character.

Large losses of fat in the form of soap in the stools, and the production of hard putty-like masses of evacuation, are characteristic of certain putrefactions that appear in the gut; these are not dependent on a diminution of the digestive power but on the presence of a preponderance of certain putrefactive bacteria.

Intolerance for carbohydrates is largely a quantitative matter. The hygroscopic power of the carbohydrates, sugars especially, comes into play when the concentration of the solution used is too great, with the result that irritation of the gastric mucosa is produced. *Distress and sometimes vomiting* occurs if the stomach be dilated or if the pyloric reflex be brought vigorously into action. Certain bacteria that feed on carbohydrates may accumulate in the stomach and produce lactic and allied acids, and thus cause distress with subsequent vomiting. Carbohydrates, however, are much more prone to produce ill effects through their action in the intestine than in the stomach. The disaccharides and polysaccharides often are carried unutilized into the lower levels of the ileum where they may encounter a rich growth of fermentative bacteria, with the result that they are split rapidly and an excess of lactic acid is produced. This condition is more properly the result of bacterial action than of lowered digestive capacity.

The monosaccharides are absorbed high up in the gut and often when there is sugar intolerance with a fermentative diarrhea from the use of milk sugar, cane sugar, starches, or dextrin, maltose and glucose, (in the form of corn syrup) are utilizable.

The only purely *digestive disturbance brought about by proteins* is the result of the curdling produced by rennin on some milks. Why one milk should clot in large masses and another in small flocculi is not clearly understood but is probably the result of some colloidal peculiarity of the casein. Occasionally large curds form in the stomach; these are really casts of the pylorus. Children have been known to strangle while vomiting such cheese-like masses. More often, the gastric muscles force these large masses past the pyloric sphincter into the duodenum where they are attacked by the pancreatic ferments but are imperfectly dissolved. They may act as foreign bodies in the intestine, and produce abdominal pain; as a result of incomplete digestion, they appear in the

stools as large bean-like masses of curd. This form of protein indigestion is not very rare but it can be prevented easily if boiled milk is used in the feeding mixture.

Objections to the overuse of protein are valid, but they do not run in the field of lowered digestive capacity. Rather, they are revealed by disturbances of metabolism which may be due either to the protein itself, or to absorption of protein-split products engendered by the action of proteolytic flora on the excess of protein in the intestine. The fact that protein-milk and lactated-milk can be taken in large quantities by children whose tolerance for carbohydrates and fats is low, is a clinical fact of great importance for this type of child.

The *treatment* of the digestive disturbances due to lowered digestive capacity of any individual element of the food is a matter that may sometimes test the clinical ability of the physician to the utmost. In these circumstances, a knowledge of the percentage composition of the feeding mixtures renders it possible to control the proportion of foods given and is therefore of paramount importance. Ability to vary the fat content from day to day and to change the amount of carbohydrates at will may make all the difference between success and failure in feeding a child with a lowered digestive power.

When the vomiting of *fats* and *fatty acids* and the presence of gastric discomfort are dominant, the indications are to begin by feeding skimmed milk mixtures. At times even the small amount of fat that has been left in the milk after skimming is badly tolerated. In these circumstances, buttermilk or lactated skim milk is available and almost always useful. Under these conditions too, one of the proprietary foods such as Nestles' or Allenbury's, or malted milk, any of which contain proportionately little milk-fat or milk, and which are readily prepared, may provide a useful temporary feeding. Due care must be taken so far as possible to insure an adequate energy intake for these children, although when beginning treatment, this is of less importance than it is to assure a full fluid intake. Very often feeding through one or two days with a 7 per cent to 10 per cent sugar solution without any added milk or dextrins, allows the stomach to rest and to empty itself. While it is getting rid of the accumulation of fatty acids and mucus, this semistarvation allows time for the irritation of the gastric mucous membrane to subside. Before initiating such a period of treatment, a single stomach washing with a 2 per cent sodium bicarbonate solution will be

of great value. Following a day or two of milk deprivation, the addition of fat in very small quantities may be undertaken. It is best to begin with a concentration not to exceed ¼ to ½ of 1 per cent and to increase this concentration day by day with great care. The fact that the top 6 ounces of a quart of 4 per cent milk that has stood until the cream is well risen, has a fat content of about 16 per cent, facilitates the computation and control of fat in a feeding mixture. One-fourth ounce of the top 6 ounces will add ¼ per cent fat to the pint of mixture; ½ ounce adds ½ per cent; 1 ounce adds 1 per cent; 2 ounces adds 2 per cent, and so on. Of course, care must be taken that the 6 ounces are removed when the cream is well risen and that it be thoroughly stirred before adding it to the mixture. It is as easy to enrich a 20 or 40 ounce mixture as it is one of 16 or 32 ounces in preparing the feedings, because the top 5 ounces of a quart of milk contains an approximate 20 per cent fat; so that if 1 ounce of this be added to 19 ounces of diluent, a 20 ounce mixture which will contain 1 per cent of fat will be the result. In most cases of lowered digestive capacity for fat, if this method is used, the patient can be brought in the course of 3 or 4 weeks, to a point where he can tolerate mixtures containing 2½ per cent to 3 per cent fat, and it is desirable wherever possible, that the ration should contain at least 2 per cent or 3 per cent fat. A growing knowledge of the importance of fat-soluble vitamines in the diet emphasizes this need.

In those cases where it is impossible to develop a tolerance for moderate amounts of bovine fat, we can turn to the wet nurse where one is obtainable. Failing her, we are forced to depend for caloric intake on skim milk mixtures poor in fat, enriched with dextrins and starches, or upon mixtures of sweetened condensed milks. If these milks are diluted 1 to 8, about the highest concentration at which they can be used, the resulting feeding mixture will contain 1 per cent of fat. As a matter of fact, the dilutions possible to use are more often 1 to 16 or 1 to 12, in which case the fat percentage will run ½ per cent to ¾ per cent of fat.

One of the commonly urged objections to sweetened condensed milk, that it contains overlarge quantities of carbohydrate, is not valid. If the highest usable concentration, 1 to 8 is given, the resulting carbohydrate percentage will be not over 5¼ per cent and if the food is low in fat, children tolerate a very much higher carbohydrate percentage than this, unless they have acquired a preponder-

ance of fermentative bacteria in the intestine. It is a constant clinical experience to meet babies who are being fed on a dilution of condensed milk with the addition of a cereal and who are apparently doing well. Those who have followed many of these babies through their childhood have rarely seen any failures of nutrition which could be attributed justly to this type of feeding. Certainly, rickets is not proportionately more frequent among children who have been fed on condensed milk than it is among those fed on approved formulas. However, it must be said that children who are given condensed milk are also usually those who are fed early on a mixed diet, and such a diet rapidly balances the deficiencies of the earlier feeding. It seems probable that the prejudice against condensed milk as a food for infants arises from the fact that because it is so simple to use it becomes the food of choice among lazy, shiftless, and ignorant people, and that the results of ignorance and dirt, expressed in malnutrition and poor development, are unjustly attributed to the food.

The pale, flabby baby that is quoted as a reproach to condensed milk as a food, often acquired his pallor and flabbiness from faults of regimen and an absence of proper diluents in the feeding mixtures. Overfeeding will and does often produce this type of malnutrition even when varied dietaries, many of them containing certified milk have been used. There is no reason to urge condensed milk as a staple basic diet for infants, but under many circumstances, therapeutic and sociological, canned milk becomes a valuable aid to the practicing physician. Packed by a firm of good reputation, it is nearer sterility than even certified milk; it is of uniform composition, and it is available in many places where clean dairy milk cannot be had. It is convenient to use and not expensive, and unquestionably the process of homogenization and concentration with sugar through which the milk passes, renders it more utilizable for certain children with weak digestions than any other mixture that has been devised.

Of course the use of cooked milk mixtures, whether they be made from boiled fresh milk or condensed milk, demand the inclusion of antiscorbutics in the dietary. When bovine fat is not tolerated, it is a fortunate thing that almost always cod liver oil will be accepted without trouble; and it is wise to add 4 or 5 drams a day of a 33 per cent emulsion of this oil to the mixture. It is also desirable to use the vegetable decoctions

instead of water for diluting the milk. In passing, it is well to note that certain carrots and potatoes may be poisonous to babies. Sometimes parts of these vegetables which remain above ground during growth exposed to the sun, acquire a dark green pigment. Such pigmented vegetables should be rejected for human food.

The unsweetened condensed milks are also convenient and valuable. Diluted with equal parts of water, they can replace boiled fresh milk in most formulas and they are equally valuable except for the fact that there is no way other than dilution of altering the fat composition of the mixtures prepared from them. They should not be fed without the simultaneous use of the antiscorbutics. They are less valuable in the feeding of children who have suffered from digestive incapacities for fat than are the sweetened condensed milks or simple skim milk mixtures. They require the addition of sugar, dextrins or starches and the process of condensation seems to make them a little more tolerable to some stomachs than simple boiled or raw milk. This increased tolerance probably follows because of the homogenization of the milk which renders the fat emulsion more uniform and the fat more readily digestible. Condensation brings about no change in the chemical or physical nature of the proteins although its physical state after precipitation is altered.

The *treatment of carbohydrate intolerance* in infants, when the intolerance is expressed by watery eructations, and by reddening of the tongue and mouth, is best begun by the entire withdrawal of carbohydrates for a day or two. If there is much discomfort, a single stomach washing with a 2 per cent sodium bicarbonate solution will often restore the balance quickly. When the intestinal symptoms of overfeeding with carbohydrates—diarrhea, proctitis and excoriated buttocks—are dominant, the withdrawal of carbohydrates for 48 hours and the feeding of protein-milk with slight sugar addition will restore a child to the normal state unless through neglect, the condition has been prolonged over many weeks. When proctitis or rectal discomfort is great, one or two colonic flushings with a 5 per cent sodium bicarbonate solution will ease the distress. The excoriated buttocks are best treated by exposure to the air as detailed in the chapter on Methods.

The *treatment* of lowered capacity to digest proteins rests upon the replacement of raw milk by boiled or condensed milk or in

some cases, upon the substitution of one of the foods containing a dried milk. The condition is rare except in the mild form evidenced by bean-like masses of curd in the stools or the vomiting of large coagula.

Digestive incapacity due to the presence of concomitant diseases such as pneumonia, bronchitis, otitis or disturbances of the circulatory apparatus, demand a treatment different in no way from that advised for the digestive incapacities of a congenital nature. During any illness, the child's digestive power is depressed and most often the ability to split and absorb fat is the function most damaged. The measures already outlined hold under these circumstances. The fat should be withdrawn for a short period and be given only as the digestive powers improve.

The great need of a sick child is for fluid and for easily utilizable carbohydrates, provided always that the intestine is not overrun with sugar-splitting bacteria. The simple sugar solutions and cereal decoctions are available. In the physician's judgment the moderate addition of skimmed or condensed milk may be made as needed. The citrates and malates of fruit juices are valuable dietetic adjuvants, and in older children, they can be used in reasonable quantities for their diuretic and diaphoretic effect as well as for their power of alkalinizing the tissues. There is a prejudice against the use of acid fruits because of a fear that they will produce acidosis. This view should be combated as it rests on an oversight of the fact that the citrate and malate groups of organic acids neutralize acidity and tend to render the urine alkaline.

Nutritional Disorders Due to the Action of Varying Intestinal Flora

The classification of Finkelstein and his followers which divides the nutritional disorders into balance disturbances, dyspepsia, and intoxications, has been useful, but it is complex. From time to time, its originator has shifted his ground and changed his teachings, while many of his followers have failed to keep pace with him. This has led to much contradictory teaching on the subject.

"**Balance disturbance,**" (the evidence of which is loss of weight, inability to utilize food at a normal intake, loss of turgor and muscle tone, impaired color, restlessness, disturbed sleep, occasional vomiting and oscillating temperature line), is in reality a complex often resulting from a feeding mixture too low

in carbohydrate and too high in fat and protein, especially the latter. Such a food provides an intestinal content encouraging to the growth of putrefactive organisms whose activities lead to the production of soap stools, in hard, putty-like masses difficult to evacuate. When fat is split and absorbed imperfectly, these soap stools are produced by a combination of the fatty acids with calcium and magnesium derived from the food or abstracted from the intestinal circulation. The vomiting, which is rarely extreme in this condition, is due to a mild pylorospasm.

One fact that is emphasized as part of the clinical picture, is the ammoniacal diaper. The odor of ammonia in the diaper has nothing whatever to do with the ammonia coefficient in the urine. It is a metabolic impossibility that ammonia should be excreted uncombined, and the ammonia odor about such children, is due to the fact that certain urea splitting bacteria inhabit their intestinal tracts. These contaminate the diapers and urinals and come into contact with the urea of the urine, break it down and thus produce this ammoniacal odor. Faber was able experimentally to produce an ammoniacal odor by placing the feces of affected children into contact with normal urine. Cook, working independently, after ingenious experimentation, completely proved the point and succeeded in isolating the urea splitting organism. He devised a therapeutic measure of controlling the odor; he has the napkins soaked, just before the drying process, in a saturate solution of boric acid or in a weak bichloride of mercury solution. The boric acid solution is preferable because of the ever present danger of bichloride poisoning in a household containing children.

It would seem more fundamental to displace the urea splitters by increasing the carbohydrate and lessening the protein in the diet, as above indicated.

The position is taken by some writers that the putrefactive bacteria are a result and not a cause of "balance disturbance." As a matter of fact, if food is so modified that the intestinal flora change from a putrefactive to a well balanced or dominantly fermentative flora, the various morbid conditions grouped under this heading abate and the child begins to improve; whenever a child does begin to improve, it is perfectly demonstrable that the flora has changed from one type to the other.

Under the term "**decomposition,**" the proponents of the Finkelstein classification describe what in reality is a severer development of the "balance disturbance." In what is called "decomposition," starvation and lessened blood volume are added to the original dis-

turbance with the result that vomiting, instead of being occasional and slight, becomes a frequent though rarely a distressing symptom. The slight variations in temperature range, give way to a hypothermia which may be as low as 95°. The lowered blood volume is expressed in circulatory disturbances such as a slow, weak pulse, and a slight but progressive accumulation of acid bodies in the blood which brings about rapid breathing; in the severer cases this breathing becomes irregular. Loss in weight becomes extreme, and the child develops inability to digest and to utilize food of a composition that should be perfectly adapted to its nutritional needs. As a result the "paradoxical reaction" follows. This is indicated by the fact that the child's weight decreases as its ration is increased. The examination of the urine will show that secretion to be concentrated and to contain albumin, casts, white blood cells and sometimes red blood cells; on occasions, sugar may be found.

These severer symptoms and more profound metabolic disturbances have followed the ingestion of an ill-balanced diet in the presence of a preponderantly proteolytic flora. They are primarily due to altered water metabolism and the proof of this is that in many instances a restitution of water to the tissues, either by intravenous, intraperitoneal or subcutaneous injection, will permit nutritional processes to reestablish themselves if the carbohydrate intake is properly balanced against the protein and fat of the food. The test of a proper balance will be the appearance of a mildly acid, soft stool replacing the hard, putty-like, stinking, putrefactive masses that were formerly present.

With the dilution of the body fluids, irritation of the kidneys is mitigated and in a short time, casts and albumin will disappear from the urine; the circulation is restored with an improvement in color and the pulse becomes more normal. Following these changes, the need for forced ventilation of the lungs disappears and the respirations become slower and more regular. Under some circumstances, in the severer cases, it is necessary to add a colloid to the fluid injected into the blood stream in order to prevent its escape by way of the kidney or into the tissues. The best colloid for this purpose is glucose in slightly hypertonic solution, although acacia or gelatin may be used. (See Methods.)

"**Dyspepsia**" is the term that has been chosen to represent a group of clinical manifestations, the chief of which is diarrhea. The symptomatology given as representative of this condition is that of a gastroenteritis with great increase in the frequency of the stools which are liquid and green, and which may be either

extremely fluid, or semifluid, mixed with large quantities of mucus. Almost any etiologic factor may bring about this state of affairs; overfeeding, underfeeding, the use of raw milk, the excess of any of the individual elements of the food, infected milk, bacterial infection either in the intestinal tract or outside of it—any or all of these, according to those who use this method of classification, may be etiologic. As a matter of fact, these symptoms in the great majority of cases, arise from an excessive carbohydrate intake with lowered tolerance and digestive capacity together with the presence in the intestine of a preponderant saccharolytic (fermentative) flora. Encouraged by the presence of unsplit disaccharides or polysaccharides, the products of bacterial growth produce an excessively acid stool. The development of a diarrhea follows with a diversion of body fluids and of salts from the blood stream into the intestinal tract; and if the process runs on unchecked, the water and salt losses result in blood concentration and the development of metabolic incapacity which can be remedied only by the restoration of water to the tissues.

The *treatment* is through diet and by the restoration of fluid to the tissues. For a period of 24 to 36 hours, carbohydrate should be entirely withdrawn. In the graver cases, water or tea alone should be used for 12 to 24 hours. After this period, protein-milk should be used. In the moderately severe cases, protein-milk with 1 per cent malt-dextrin or dextrin (corn syrup), may be given. The food of choice in these cases is protein-milk (see Formulas), but it should be given at first in limited quantity. A 15-pound baby should have 15 to 20 ounces of protein-milk during the first few days. For 24 to 48 hours, corn syrup or malt-dextrin should be added in the proportion of $\frac{1}{8}$ ounce to 20 ounces. The carbohydrate addition should be increased $\frac{1}{4}$ ounce daily, until an added 4 per cent or 5 per cent has been attained. The final sugar content of the food is to be determined by the clinical conditions, especially by the character of the stools. The recurrence of diarrhea may render it necessary to withdraw a part or all of the carbohydrate for a day or two and to return to the method of gradual daily increase of this substance. If improvement is prompt and continuous, the amount of protein-milk fed daily may be increased gradually, until the baby is getting 6 or 7 ounces 5 times a day, an amount of food, which together with the added carbohydrate, will meet its energy needs. When the stools become normal and the condition of the child is

satisfactory, protein-milk may be withdrawn. This is best done by at first substituting for 1 feeding of protein-milk a normal formula. In the course of 6 or 7 days, all the protein milk should have been withdrawn and replaced by rational feedings. However, if a tendency to a frequency and looseness of the stools persists, it may be well to continue the feeding of 1 or 2 bottles of protein-milk for weeks or even for months. The slighter cases will respond to the withdrawal of carbohydrate and the feeding of boiled milk. For older infants, it is better not to dilute the milk but to reduce the fat content to 1 per cent or 2 per cent by removing the top 12 or 6 ounces. Infants under three months tolerate the boiled milk better if it is diluted with one-fourth of its volume in water. In using the concentrated, boiled milks, smaller feedings are given and water must be fed in the intervals between meals.

If the diarrhea has persisted for some time and if the child is much dehydrated, it will be necessary to initiate the treatment by the subcutaneous or intraperitoneal injection of water. (See Methods.) In the milder cases, sufficient water can be given by mouth.

There are a few cases, in which the acidity of the stools comes from an excess of butyric acid (easily recognizable by its odor), that fall in this category; in these the evidence points to a high putrefactive flora as causative. When we are confronted with one of these cases, the indication is to withdraw milk in all its forms and to feed 7 per cent to 10 per cent lactose or maltdextrin solution, preferably the former. As this solution leaves the stomach quickly and is rapidly absorbed, it should be fed every two hours in liberal quantities. Twenty-four hours later, the proportion of sugar in the solution may be reduced to 5 per cent, and a 5 per cent cereal decoction made of barley, wheat and rice flour added. For nurslings, this feeding may be continued for a week or ten days with good effect. There is advantage in adding a small piece of butter to each bottle of food just before serving it. After the butter is added, the mixture should be thoroughly shaken.

These patients often do well on the thick formula discussed in the chapter on Vomiting. Usually after three weeks of carbohydrate feeding, milk may be added to the dietary. It is best to begin by using a small quantity of top milk, 1 ounce of the top 10, to a quart of gruel, increasing 1 ounce every second day until the child takes 8 to 10 ounces of the top 16, in a 32 ounce mixture.

Variations in Metabolic Power.—Many of the reasons offered

as explanatory of the metabolic disturbances of infants are pure assumption. However, thanks to Henderson, Howland, Marriott and others, we are able to look with some clearness into the changes which lead to the metabolic disturbance known as *acidosis*. We can appraise with some precision, its causes and its results and provide preventatives and remedies. The results of changes in blood volume have been removed from the field of conjecture. Much is attributed to changes in the balance of the different minerals that enter into the body composition, but here knowledge is far from sure. The influence of the internal secretions of various essential glands on metabolic processes is much studied, thought of, and written about. Except as to the effects of the thyroid, normal or abnormal, there is no surety as yet in our knowledge.

Alterations in fat metabolism may lead to the production of abnormal acids in the blood or to the production of acetone bodies, although these events are unlikely to happen except during carbohydrate starvation and in diabetic patients.

The clinical picture described as "**acute alimentary intoxication**" by the German observers, is one essentially accompanied by changes in the water metabolism of the child and the events that lead to this disturbance of water metabolism may be vomiting and diarrhea that result from the ingestion of improper or infected food, a disturbed floral balance in the intestine, an active invasion of the intestinal mucosa by pathologic organisms or any disturbances secondary to infections in other parts of the body. Any one of these causes, although insufficient in itself, may be reinforced by the effect of excessively high external temperature, especially when the humidity is also high. It is in this class of disturbance that unknown changes of intermediary metabolism have been most invoked as explanatory, although the concentration of the blood, with the resulting disturbances in salt balance, blood protein concentration, and the production of an acidosis, is sufficient to account for all the symptoms in many of the cases. In others, the liver may be incriminated. Although there is no proof, there are many reasons to believe that an undiscovered living virus is responsible for the pathologic changes in this organ; changes which lead to atrophy and fatty infiltration and to a destruction of the hepatic detoxicating function. It is these cases with liver damage that occur frequently in the southern parts of the United States. They are unresponsive to the best considered therapeutic measures at present available.

The symptoms that indicate alimentary intoxication are fever, vomiting, with collapse, often, but not always, diarrhea, and excessive and rapid diminution in weight due to fluid loss from the body. Forced ventilation of the lungs is a striking feature, and in the later stages or in the severe cases from the beginning, hyperpnea is extreme and respiratory irregularities may be well marked. Apathy and drowsiness are almost always dominant, although an urgent restlessness is sometimes to be observed. Ordinarily these children are inert, and when they do make an effort, their movements are slow and deliberate. The facial expression is most characteristic. The eyes are sunken, and the look is one of extreme weariness and lack of interest, quite in contrast to the anxious expression seen in severe cardiac and respiratory disturbances. The face is usually of a pale, grayish hue, the lips and tongue are dry and the mouth parched. In the terminal stages of the disorder stupor is profound. Convulsions occur sometimes, but not very frequently, except as an ante-mortem event, although twitching and muscle spasm are often encountered. There is an alimentary glycosuria in some cases, although this phenomenon is far from constant. There is always evidence of kidney irritation if urine is secreted. In some cases, renal secretion fails and the picture becomes complicated by that of uremia. The white blood cells are more or less increased in number. The refractometer shows concentration of the blood and a relative increase in its protein. In the type of case that is usually fatal, there is marked enlargement of the liver with some tenderness. An inquiry into the history of these patients will usually indicate some previous digestive disturbance or some severe prior infection, either gastrointestinal or parenteral.

The *treatment* of alimentary intoxication is simple, except in those cases in which liver degeneration is a complication, and it is usually promptly effective; it calls for a preliminary food deprivation for 16 to 24 hours together with the provision of ample fluid. This may be given in the form of weak tea or as plain water. In the severer cases, the fluid will have to be administered subcutaneously or intraperitoneally and in the most severe, intravenously. When the fontanel is open, the longitudinal sinus is the vein of choice. When the blood stream is chosen, there is advantage in using a solution containing 7 per cent of glucose. Five minims of a 1 to 3500 adrenalin solution may be added to this solution. Some authors recommend the injection of sodium bicarbonate with the glucose, but this procedure is dan-

gerous and unnecessary; the acidosis is more effectively combated simply by the restitution of a proper circulatory volume.

A single stomach washing may be of great importance when vomiting is a striking feature. The use of iced milk of magnesia in dram doses every ½ hour for 6 to 8 doses is of distinct advantage. It tends to allay vomiting and to hasten the cessation of the diarrhea.

If the child is in collapse or even if its stupor is moderate, a mustard pack is of great use in stimulating the sensorium and in increasing the force and regularity of the pulse and in steadying the respiration. There is no better stimulant than the mustard pack in this condition, but it may be reinforced by the hypodermic injection of 10 to 12 drops of camphor (10 per cent) in olive oil every 2 or 3 hours. When the weather is very hot, and the child has a fever, cold packs may be used or the child may be placed in a cool tent, such as is described in the chapter on Methods. The use of an electric fan, or failing this, hand fanning, may save the child's life by maintaining a temperature around the body low enough to encourage rapid radiation.

In cases of urgent restlessness or when diarrhea is excessive and persistent, the use of opium is indicated. The best form in which to use the drug is the deodorized tincture, (minims ½ every 3 hours for 3 doses for a 15 pound child), or morphin sulphate may be given by hypodermic injection (1/120 grain repeated at 3 hour intervals up to 1/40 grain for a 15 pound child).

The fluid (water or weak tea) should be continued in quantity following the preliminary starvation period. During the second 24 hours, food should be given in small amounts. The kind of food to be chosen will depend on the character of the stools. If, as happens in many cases, the stools are of the fermentative type—green, sour and excoriating to the buttocks, the indication will be for the use of protein-milk. The meals should be small and fairly frequent; 10 or 12 in 24 hours, each to consist of 1 or 2 ounces according to the size and weight of the child. The milk should be sweetened with saccharine and after the third day 1 per cent to 2 per cent of malt-dextrin or corn syrup may be added. Much damage can be done in this condition by withholding even small amounts of carbohydrate for too long a period.

By the third or fourth day, the stools should become at least semisolid. It is now time to increase the carbohydrate daily

until at the end of a week, 4 per cent to 5 per cent of sugar in the protein-milk mixture is being ingested. Julius H. Hess has recommended that 1 per cent dextrin, (flour ball) be used with the protein milk in the first feedings. It is best to limit the intake of protein-milk and not to give any child more than a quart or at the most 40 ounces a day. Most children will do better if their daily intake is restricted to 24 ounces of this preparation.

When the improved condition of the child warrants a more normal diet, the change must be made gradually. Daily, one bottle of a rational formula should replace a feeding of the protein-milk mixture and after another day or two, another bottle of formula is added and so on until the child is taking a formula proper for its age and weight.

In the less severe cases, it is not necessary to resort to protein-milk feeding but boiled milk, either wholly or partially skimmed may be used; as the symptoms abate, gruels of oatmeal or farina may be added in appropriate amounts. It is wise to forego the use of sugar in these solutions because the lactose present in the milk will provide ample sugar. Sometimes the amount of carbohydrate present in the milk will be found to be excessive, and a recourse must be made to feeding with protein-milk in the way suggested for the more severe cases.

When the diarrhea is of the putrid type or when there is no diarrhea and the stools are constipated, or even normal in consistency and number but very foul in odor, there is every probability that we are dealing with an intoxication, accompanied by a proteolysis in the intestine. Under such circumstances, the feeding of protein-milk will aggravate the symptoms and may lead to prolongation of the metabolic disturbance. When the evidence leads us to suspect this intestinal condition, it will be proper to choose a carbohydrate feeding and such a feeding should be followed by an amelioration of the symptoms. Where it is possible to study the intestinal flora by laboratory aid, the improvement in the clinical condition usually will be found to coincide with a change in the flora from a proteolytic to a saccharolytic predominance.

Under these circumstances, the first 24 hours' feeding should be of water or weak tea. The second 24 hours' feeding should be a 7 per cent lactose or dextri-maltose solution. In severe cases, it may be impossible to feed anything else than this sugar solution for 2 or 3 days, although it is usually possible to take the next step in the dietetic progress within 24 to 48 hours. This

consists in feeding a preparation of a cereal gruel made from farina, rice flour or oatmeal, added to a 5 per cent sugar solution. If there is no sign of acidity in the stools, or when the symptoms pass, the addition of butter, 1 teaspoonful to 8 ounces of cereal decoction, should be made. In place of this, 2 teaspoonfuls of egg yolk may be stirred in the boiling gruel. After ten days or two weeks, small amounts of milk may be added to the gruel and very slowly, through a period of 4 to 6 weeks, the child may be brought back to a normal formula in which, however, carbohydrate should be kept high. At least 7 per cent, often 10 per cent of carbohydrates in a mixture made up of sugar, dextrin and starch, will be tolerated and will prove to be of advantage.

The satisfactory nutritional results that have followed the use of thick formulas are evidence that in the past, we have been unduly fearful about feeding carbohydrates. A careful watch must be kept on the stools and if there is any sign of carbohydrate intolerance as expressed in green, acid, excoriating evacuations, the sugar and dextrin should be withdrawn and 3 per cent or 4 per cent starch alone used with the milk until the symptoms disappear.

The condition spoken of by some writers as **anorexia nervosa** is in reality an expression of chronic alimentary intoxication in which the most striking and persistent symptom is loss of appetite to a point where a child refuses to take all food and often even water. In many of the cases, the depression of metabolism which leads to the development of the clinical picture, is initiated and maintained by an infection. A chronic otitis with suppuration, a pyelocystitis, a furunculosis or some other pyogenic process may be causative. As a complication of mesenteric tuberculosis or of rickets, the condition may be perplexing. As a rule, there is no diarrhea and vomiting is rare except when a meal has been forced. The loss of weight is gradual, the expression of the face is one of mild weariness, and the skin is pale. The hemoglobin is lowered and a leucocytosis is usually present even when there is no demonstrable suppurative process. The urine is scanty and often shows hyaline and granular casts and traces of albumin. The stools are infrequent and scanty and like all starvation stools, carry a high proteolytic flora. The absolute lack of appetite and refusal of food in which the child may persist for weeks until it finally dies of starvation, is the alarming feature of the condition.

The *treatment* consists in repairing the ravages of any infection that may be present. In girl babies, pyelocystitis is the commonest infection that has to be dealt with. Chronic suppurating ears or a furunculosis are the infections that most often need to be remedied in the case of boys.

An important part of the treatment of the condition, consists of the administration of large quantities of water, by mouth in the milder cases; or by injection, subcutaneously or intraperitoneally, in the more severe. Intravenous injection is necessary in the most extreme instances of the disturbance. The feeding should be varied according to the needs of the child. Often, milk will be refused entirely when other foods may be taken.

The stomach should be washed out every 2 or 3 days with a warm solution of 2 per cent sodium bicarbonate. The bowels should be flushed daily for a few days. For this purpose a 2 quart enema of 5 per cent sodium bicarbonate is advisable. Unless there are frequent acid stools, indicating the presence of a high fermentative flora, lactose in 5 per cent to 7 per cent solution is the best form of food to use in the beginning of the treatment. The addition of orange juice or loganberry juice gives a pleasant acid flavor to the solution and sometimes it will be taken by these children when everything else is rejected. Solutions of cane sugar and other sweets are usually refused because they are cloying. Following a day or two of lactose feeding, thin gruels of oatmeal or farina or cornstarch are to be used. These should not be sweetened, and unless the child is showing some starvation edema, sodium chloride should be added until there is a distinct salt flavor to the food, as this condiment seems to stimulate both appetite and digestion under these circumstances.

After perhaps a week of exclusive carbohydrate feeding, milk may be added but very often if the attempt is made to force children to take milk, they will again refuse food, although they have been taking cereals with a fair degree of desire. It is well always to remove the fat from the milk before attempting to feed it to a case of anorexia of this or any other type. Little by little, more milk can be added and cooked with the gruels and as time passes, more and more of the upper layers of the cream may be allowed to remain for use in the child's diet.

Beef juice in dram doses may be given the child not because of its nutritional value which is very slight, but in order to stimulate the flow of the gastric juice. Fruit and vegetable juices

play an essential rôle in the diet of these patients. Either orange juice, loganberry juice or apple juice or scraped apple should be offered once a day. The cereal should be cooked in vegetable decoctions instead of plain water and for older infants, vegetable purées, carrots, spinach, lettuce, chard, endive and the white parts of cauliflower should be given, very finely puréed, in amounts of one or two tablespoonfuls a day. In the absence of other green vegetables cabbage can be used provided it is thoroughly cooked and the water changed once during the boiling. The general prejudice against cabbage as a food for older infants is not well founded, provided the vegetable is properly cooked.

Children who develop this type of anorexia are usually between 10 and 20 months of age. Care must be taken that when they return to a normal appetite and diet, they are not given more than a quart of milk a day. Often, it is of advantage to remove a part of the cream for some time. A few of these children have so well developed an aversion to ordinary milk that buttermilk, lactated milk or protein-milk may have to be used.

In those cases in which the stools are of the fermentative type, instead of beginning the treatment with sugar solution, it must be instituted by giving boiled skimmed milk or protein-milk in dilution and the carbohydrate should be added cautiously as the stools change from the sour, small, green-looking evacuations to those of more normal appearance.

In the cases of the very intense anorexia in which the child cannot be induced to take foods voluntarily, the stomach tube must be resorted to. Children have been fed by tube for two weeks or more before they made any effort to feed themselves. But if the nutrition of such infants is maintained by forced feeding, they almost always acquire a normal appearance and good health. Care must be taken not to overfeed when using gavage. The simple anorexias of this type must be carefully differentiated from the profound anorexia that may occur in cases of *pancreatic insufficiency* or as it is sometimes called, *celiac disease*, although anorexia is not a frequent symptom of this condition. (See chapter on Diarrhea.)

Physicians, especially those practicing in the urban communities, are familiar with the clinical picture of well developed **rickets.** The thickening about the epiphyses of the long bones, and about the centers of ossification of the flat bones of the vertex of the skull; the bending of the long bones that follows softening due to imperfect calcification; the lowered tone of the muscles;

the evidence of imperfect nutrition in the epithelial structures, hair, skin and teeth; the circulatory changes responsible for vascular contraction with pallor; these combined, produce one of the most typical clinical pictures to be met in the pediatric field.

Investigation of the blood in many cases shows that this tissue also suffers. Diminution in hemoglobin and changes in the red cells, while not constant, are of great frequency, and an increase of the white blood cells is the rule.

The lowered tone of the abdominal and back muscles allows the older infants to assume the characteristic pot-bellied posture. The lack of tone in the unstriped muscle of the intestine permits abdominal distention. The involuntary muscle fibers of the bronchi are also affected and probably become a factor in the production of the frequent bronchitic attacks from which rachitic children suffer. The deficient nutrition of the epithelium of the digestive and respiratory systems is also contributary to the disturbances in these tracts.

The part played by ill lighted and badly ventilated rooms in aggravating rickets, long has been recognized. However it is Alfred Hess who has given scientific explanation of the influence light waves have upon debilitated metabolism, and who has appraised their value in the treatment of rickets.

The nervous system is especially dependent on a proper protein-salt balance for its functioning. Irritability, restlessness and disturbed sleep are frequent accompaniments of the rachitic state. The most striking of its nervous manifestations is **spasmophilia,** a condition in which the peripheral nerves exhibit an exaltation which probably is dominant throughout the whole nervous system. Uncontrolled nervous discharges may occur with a resulting convulsion or when the discharge is less intense, the child may merely lose consciousness for a moment without exhibiting motor manifestations. Any group of muscles may be subject to a few twitches without the patient's consciousness. A common expression of a rachitic nervous system may be seen in *laryngismus* where spasm of the larynx arrests expiration for some seconds and breathing begins again only when carbon dioxide accumulates and stimulates the respiratory center. A great deal of breath-holding attributed to sheer naughtiness, is in reality, an expression of spasmophilia, and if the diets of children who exhibit this trait be carefully scrutinized, it usually will be found that the elements of the ration are ill-balanced. Most of the attacks that mothers call "inward spasms," and

many of the convulsions that occur between 8 and 16 months, are essentially spasmophilic in origin and can be minimized or even cured by careful attention to diet and hygiene. Calcium chloride and cod-liver oil are essential.

The essential signs of spasmophilia are the staring eye, the tetanic position of the extremities (forearms flexed upon arms, hands on forearms, fingers together, thumbs opposed to ring fingers, extended phalanges with flexion at the metacarpophalangeal joints), the longitudinal arching of bony structures of the feet and a deep antero-posterior grooving of the skin of the sole from the roots of the second toe to the middle of the instep. Chvostek's and Trousseau's signs are usually present. Hyperexcitability to electric stimulation is shown in the alteration of electric responses to the galvanic current. Any response to a current less than 5 milliamperes warrants suspicion.

Cod-liver oil and phosphorus are the only drugs of proven value in rickets, although the chlorin ion, especially in its combination with calcium is essential in the *treatment* of spasmophilia. One eight hundredth of a grain of phosphorus to the dram of 33 per cent emulsion of cod-liver oil, given to a 15 pound child 4 to 6 times a day, is usually well taken and is sufficient. The pure oil is well tolerated. Cod-liver oil has not only nutritional and vitamin value but also its high olein content dissolves soaps and fatty acids or other fats ingested and renders them more absorbable from the intestine. The fact that Howland and his co-workers found the calcium in the blood of rachitics to be considerably less than normal may have some bearing in this connection. These investigators found the chloride to be the form of calcium most effective in restoring calcium balance to the blood. If the lactate is given the dose must be about double that of chloride.

Dietetically, a well-developed case of rickets calls for a balanced diet in which milk plays a not too predominant part. Scraped meat, green vegetables, cereals, especially oatmeal and egg yolk are the staples that must be most depended upon. The amount of fluid in the diet is an important factor in the treatment of these patients, but apparently what they need more than anything else is utilizable fat and a high protein range in the ration.

An essential part of the treatment of any nutritional disorder, especially rickets, is an insistence on hygienic living conditions. The child should be kept in the sunshine and open air as much

as possible. A daily bath should be given and after the bath, the child should be rubbed in order to improve the circulation and to aid in the restoration of muscle tone. The services of a trained masseuse will be of advantage.

In addition to the highly beneficial effects of heliotherapy (see page 576) in the treatment of rickets and other nutritional disorders, the quartz lamp has recently come into use. Under conditions where sun therapy cannot be employed, it is of particular value. Metabolism stimulation is its outstanding virtue.

The prevention of *deformities* and their *treatment* is a matter for consideration by an orthopedist. However, the extraordinary power of normal growth to correct rachitic deformities must never be overlooked. Many young children with mild deformities that would be naturally corrected by the growth of the next year or two, are unnecessarily subjected to plaster-casts and corrective apparatus. This is especially true of deformities about the lower ends of the tibias with eversion of the feet and flat arches. These conditions are self-corrected if attention is given to a proper shoeing and to appropriate exercises for strengthening the muscles of the feet and legs, if at the same time a rational diet is insisted upon.

In those cases in which digestive disturbances, diarrhea and flatulence, are dominant features, the dietetic treatment will usually be efficient. Here, as in all other intestinal disturbances, the type of intestinal flora will help determine the best make-up for a dietary. If flatulence is due to the presence of the spore-bearing anerobes, as it often is, the starvation treatment for their eradication outlined elsewhere should be instituted early in the treatment. For years, English physicians have used gray powder, (mercury and chalk), in ¼ grain doses 3 or 4 times daily with advantage, in this complication. It is good practice to combine this mixture with taka-diastase, grains 2; saccharated carbonate of iron, grains 2; and powdered calcium carbonate, grains 2; the latter not to supply calcium to the body but with the idea that its presence in the duodenum improves intestinal digestion.

The respiratory complications are to be dealt with as any other subacute bronchitis is treated. Mustard packs are advantageous and the mild expectorants may be used if desired. Creosote given in minim doses in milk is often of service. The syrup of hydriodic acid in 5 to 10 minim doses carried in syrup of althea or wild cherry is a valuable aid in terminating a lingering subacute bronchitis in which secretion is viscid.

Careful scrutiny of the history of any instance of fully developed rickets will usually reveal the fact that there was a prodromal stage in which the child suffered from a nutritional disturbance with symptoms quite different from those generally accepted as of rickets. The striking features of this nutritional disorder are pallor, head-sweating and motor restlessness. The child constantly rubs its head on the pillow until the hair is worn off the occiput. The appetite is disturbed—sometimes it is voracious and sometimes quite the opposite. The tissue-turgor is lost and the muscle tone diminished. The child ceases to gain in weight or gains very irregularly. The temperature line is apt to be irregular, ranging a degree above and below the normal. Irritability is often a feature. The stools in most instances are constipated, putty-like masses which consist largely of soaps. The diapers frequently are yellow stained and the ammoniacal odor is marked. In other words, the clinical picture is that of the nutritional disorder which has been called "balance disturbance."

By no means, all of the children who suffer in this way go on to the development of florid rickets. Many find health through a corrected regimen and the rachitic state is averted, but no case that develops rickets escapes this preliminary stage of perverted nutrition. Balance disturbance is accompanied by a high proteolytic flora in the intestine. By appropriate changes in the diet, (the reduction of milk when it has been fed in excess, and the establishment of a well-balanced mixed diet as early as the seventh or eighth month), a change in the intestinal flora can be produced.

There is no more typical clinical picture during the first two years than that of **infantile scurvy**, and yet many cases of this disorder are allowed to pass undiagnosed. Almost the earliest expression of the disease is generalized tenderness. The child resents being picked up and handled and this may be the only sign for several weeks. Therefore, in the presence of such a symptom, the physician should consider the possibility of developing scurvy and scrutinize the feeding carefully.

Since pasteurization of milk has become popular and since it is found to be wiser in general to use boiled milk, in spite of the general opinion that the disease is rare, the danger of the development of scurvy is always with us. As early as the second or third month, a few drops of orange juice, well diluted, should be added to the dietary, once or twice a day. Thanks to Alfred Hess, we know that when we are unable to obtain oranges, we may make recourse to canned tomato and that its juice may be given in place of orange juice with satisfactory effect.

After tenderness, the earliest sign of the disease may be found on an examination of the mouth. The gums are spongy, swollen, purple in color, ecchymotic in spots and very tender. The child may refuse the bottle, and in the hands of one of those mistaken physicians who lance gums, it may have been subjected to gingival incision, much to its added discomfort.

Coincident with the changes in the mouth, ready bruising of the skin is apt to develop with ecchymotic spots showing here and there, especially where the clothing may be tight or where the fingers of the attendants grasp the child in lifting it. The body becomes increasingly tender and movement is strongly resented. The smaller blood vessels about the epiphyses give way and some subperiosteal and epiphyseal hemorrhage takes place, with the result that swelling about these regions becomes evident. The tenderness is further aggravated in this way and the child protects the affected limb by preserving an immobility so complete as to mimic paralysis; and as a result the descriptive term "scorbutic pseudoparalysis" has been widely adopted. It is usually a leg that is so affected. The lower epiphysis of the femur is the most common site for such a hemorrhage, although hemorrhages at the upper and lower epiphyses of the tibia and at the upper epiphysis of the femur are not infrequent. Occasionally, the upper epiphysis of the humerus will be affected with the result that the arm hangs in a position of palsy. Hemorrhage may occur in the periosteum of other bones—behind the orbit, over the zygoma or along the mandible. Bleeding along the spines with subsequent swelling and tenderness may mimic acute Pott's disease. Bleeding may occur elsewhere than in the cutaneous and periosteal regions and the appearance of blood in the stools or urine need not surprise the observer.

The early or "fruste" form of scorbutus which should be recognized, is merely an expression of the earlier signs in a case of scurvy which develops slowly. The symptoms are confined to generalized tenderness, palor, anorexia, ready bruising, and slight sponginess about the gums. The later feature, however, may be absent. Sometimes the only indication of the disease is the generalized tenderness. It is important to recognize these signs in order that the patient may be saved from the full development of florid scurvy. Many of the cases with early manifestations of scurvy never reach the physician, because the disease oftenest occurs during the last half of the first year or the first half of the second, when cure is accidentally effected by

the mixed diet which most children are given at this time of life.

The *treatment* of scurvy is simple and the cure amazingly prompt. It depends upon the addition of an antiscorbutic, preferably orange juice to the dietary. This should be given diluted or undiluted in ½ ounce doses, once or twice a day. Within 2 or 3 days, there is a marked change in the condition of the child. In place of the fretful, peevish patient, there is a bright, sunny one, and within ten days or two weeks the symptoms have entirely disappeared. Tomato juice, either fresh or canned, is equally valuable as an antiscorbutic, although not so palatable as orange juice. For older infants, potato cream, first advised by Still is an excellent remedy. In order to prepare it, a potato is steamed or boiled in the skin. If it is boiled, it should be dried in the oven until the skin bursts. The thin skin is then peeled off and the outer part of the pulp is to be used because it contains antiscorbutic qualities. It should be creamed with a little raw milk and fed with a spoon. The purées of green vegetables, fresh if possible, or canned when the fresh are out of season, are always valuable additions to the dietary of a child past the seventh or eighth month. Some authorities believe that the milk given scorbutic children should be uncooked and this belief seems reasonable although when the antiscorbutic foods are added to the dietary, cooked milk suffices and it is better to boil the milk if there is any doubt about its cleanliness.

The occasional appearance of **glycosuria** in infancy is not an extraordinary event. In earlier infancy when children are largely milk fed, sugar in appreciable quantities may often be discovered in the urine. This may be so when there is any intestinal disturbance, but it is more likely to happen in the presence of that form which we call alimentary intoxication. This intoxication is supposed to represent a lowered metabolic tolerance, but as a matter of fact, it is probably accompanied by some damage to the intestinal epithelium which allows lactose or other sugars to pass into the circulation before they are properly split in the intestines. These occasional and transient glycosurias of course, bear no relation to true diabetes in which the amount of sugar excreted may be greater than the amount of ingested carbohydrate. Nevertheless, any infant whose urine shows the presence of sugar on repeated examinations, should be kept under careful observation.

It is very unusual, however, to find a diabetic patient whose

age is less than 2 years, although infants even in the first month of life have had the disease. When the malady does develop in the first year, it is apt to run rapidly to a fatal issue and the whole course of the disease including the terminal coma may not exceed a month or six weeks. In later infancy, toward the end of the first year and during the second, this fulminant type is not likely to be encountered and here a period of malaise and loss of weight may precede by some weeks or months, the discovery that the urine constantly contains sugar.

The familial character of the disease is one that is to be kept in mind and the urines of children whose parents or grandparents are diabetic, should be searched at frequent intervals for the presence of sugar. Among children of such parentage, it has been shown that occasional glycosuria is very frequent, and that by dietetic training, carbohydrate tolerance can be increased and a possible diabetes forestalled.

The symptoms that reveal the fully developed case of diabetes in infancy are, most strikingly the thirst and the polyuria; accompanying these, there is listlessness, due to loss of muscular tone, and malaise. At the same time, the skin becomes dry and the tongue and mouth take on a peculiar red, dry, glossy appearance. Among these children, digestive disturbances are common. Attacks of diarrhea, alternating with constipation occur. The dry, nonresistant skin becomes the site of coccal infections. With these complications, and sometimes even without them, moderate degrees of elevation of temperature are recorded by the thermometer.

In the well-developed cases, where the alkali reserve has run low, the child is always on the edge of acidosis and sooner or later, death occurs in the coma characteristic of acidemia. The first sign of the oncoming coma is the increased effort at ventilation of the lungs; later urgent air hunger develops—a distressing picture once seen, never to be forgotten. Drowsiness is progressive until consciousness is finally lost.

The urinary findings and the blood picture seen during these stages are similar to those seen in like conditions with adults; the blood shows an accumulation of acetone bodies with a diminution of alkali reserve; and the urine is characterized by the presence of diacetic and oxybutyric acid and an increase of its total acidity and its ammonia content.

The principles of *treatment* depart in no way from those laid down for the treatment of adult cases. Theoretically, dietetic

restriction for a short period, and the feeding of carbohydrates and fat within the limits of tolerance in an effort to increase toleration by very slow and guarded increases, are to be advised. However, in practice, especially with very young babies who are still dependent on liquid forms of nourishment, the application of these principles presents great difficulty. Little children do not bear extended periods of starvation well but almost any infant can be maintained without food for 48 hours provided water is given in adequate quantities. If in a diabetic baby such a period of water is instituted, it is good practice to add alkali in the form of bicarbonate of soda, so that a child weighing 15 pounds gets 30 to 60 grains in 24 hours.

In dealing with these children, the ease with which starvation acidosis is developed must always be remembered. For this reason, the carbohydrate ration can never be made relatively so restricted as is possible with older patients. The best that can be done is to use formulas in which the protein is very high. The well washed milk curd, (see protein-milk in Methods), may very well form the basis of such formulas, and the carbohydrate additions are to be made preferably with oatmeal, without added sugar and this cereal should be restricted in amount to 2 per cent or 3 per cent of the food mixture. For example, a formula might well be made up of the curd from two quarts of milk, one fat free and the other unskimmed. If the child shows any evidence of acidosis, the further reduction of fat may be made from time to time. This curd is washed as directed for the making of protein-milk, and is pushed through the sieve with water enough added to make 30 ounces. One ounce of oatmeal that has been soaked overnight in 12 ounces of water, is boiled from 1 to 2 hours and strained through a fine seive, put into the milk curd and sufficient water added to bring the completed mixture up to 36 ounces. Six or seven ounces of this mixture may be given 5 times in 24 hours to a child of 6 or 7 months weighing from 12 to 16 pounds. A daily portion of ½ ounce of orange juice should be fed these children for its antiscorbutic effect. The small amount of sugar ingested in this way is of no consequence and the contained vitamines are essential to the child's well being.

When diabetic children have passed the bottle period, it is possible more nearly to approximate their diets to those devised for adults suffering with this malady. The 5 per cent carbohydrate-containing vegetables become available. However, it is still necessary to remember the need of the young organism

for carbohydrate and overzeal in attempting to rid the urine of small amounts of sugar is certain to get the therapeutist into difficulty. Infants at this age do much better and tolerate the amounts of fat essential to their well being better, when carbohydrate is allowed in rather more generous amounts than the degree of sugar excretion would suggest.

Joslin is of the opinion that under the modern dietetic method of treatment, children have ceased to be such bad risks as they formerly were, and that the treatment becomes hopeful. Referring to older children, he makes the statement that a rigid dietetic regimen is more comforting to the child than one that allows freedom in eating and that restriction is effective in prolonging life. A diet arranged for a diabetic child in its second year should call for about one and one-half grams of protein per pound per day. The carbohydrate must be kept low and the fat restricted but not eliminated, for the fat-soluble vitamines are essential to proper growth during infancy and childhood. To supply a sufficiency of fat without developing an acidosis will often test the skill of the clinician who will have frequently to decide whether or not it is possible to ignore small percentages of sugar in the urine and to increase the carbohydrate in order to improve fat utilization.

In the presence of a case of diabetes at any age, the physician is referred for detailed management to Joslin's "Treatment of Diabetes Mellitus" and to recent monographs on insulin therapy.

The recent development of insulin by Banting, Best and Macleod promises to make diabetes more amenable to treatment. The fact that so far this derivative of the islands of Langerhans can be administered only by workers of special training, limits its usefulness. However, the power to rescue apparently moribund children from the coma of diabetic acidosis is dramatic in the extreme.

The susceptibility of infants, with nutritional disturbances, to sudden *edema* is well known. Such an edema may be massive and extensive enough to produce anasarca. In many of the cases so affected a consideration of the diet will reveal the fact that the child is receiving an insufficient protein ration with a high salt intake. Attempts have been made to fix responsibility for the edema on an excessive carbohydrate ingestion. It is true that edema does sometimes appear when too much carbohydrate is given but only if the protein and fat of the food are kept low and the minerals high. A starvation diet (such as salted barley water or whey without addition) if given exclusively and per-

sisted in over any lengthy period will certainly lead to the production of edema. It is starvation following the inability to digest and utilize fat and protein that brings about the rarely occurring appearance of edema in pancreatic insufficiency and in tuberculosis of the peritoneum. The dropsy of the peritoneum in the latter affection is of course of other origin and depends on interference with the lymphatic system and a diminution in its powers of absorption.

The rare instances of *congenital painless edema* of the legs that have come to notice are expressions of a familial affection. The patient may be born edematous or the first appearance of the fluid accumulation may be delayed until late infancy or childhood. The disease persists throughout life and seems to have little or no effect on the general health of the patient. Not all cases of congenital edema, however, are of this type. In some, no evidence of the disorder in any related individual can be elicited. The edema may be symmetrically distributed in the lower extremities in the familial form of the disease, or it may appear in any part of the body without regularity of distribution. In either case the edema is firm and brawny and it pits on pressure. A striking feature when only one limb is affected is the tendency for the diseased arm or leg to outgrow its normal fellow. No treatment in any way influences the course of the disease, but massage and the wearing of supporting bandages contribute something to the patient's comfort.

Certain stillborn infants seem to have succumbed because of a fetal dropsy, and the condition known as *edema neonatorum* may appear in an asthenic infant born alive. Such an infant rarely survives more than a day or two because its breathing is inefficient. This respiratory inefficiency may exist because of a failure of the medullary centers to function properly or because atelectasis persists. The treatment must be directed to arousing the medullary centers that control respiration and circulation by the application of heat in the form of hot baths, by skin stimulation and by the injection of drugs such as alcohol, atropin or strychnin in appropriate doses.

Occasionally a mother who is the victim of the *toxic edema* of pregnancy gives birth to an edematous baby whose edema arises directly from its mother's poisoning. Such a child readily loses its edema if it is put for one or two days on a restricted fluid intake. The ingestion of 30 or 40 grains of magnesium sulphate in divided doses given at 2 or 3 hour intervals usually is effective

in clearing the retained fluid from the baby's body. It is important to withhold the mother's breast from such a baby as the ingestion of her milk may prove fatal to the child.

Edema as such, needs no *treatment*. Institution of a properly balanced diet, if it is possible for the child to digest and utilize it, will be followed by a disappearance of this symptom of vasomotor instability.

Malnutritions in which tetany develops and those due to congenital syphilis are sometimes accompanied by edema. Likewise the vascular disturbances that occur with anemia and with the blood dyscrasias are productive of conditions that permit serum to pass from the vessels into the extravascular tissues, a train of events productive of edema. The amelioration of such a symptomatic disturbance must of necessity wait upon effective treatment of the underlying disease or dyscrasia.

Ordinary *urticarial lesions* are smaller localized areas of edema. The causes that produce ordinary urticaria are sometimes effective in developing more extensive areas of local edema. The resulting appearances are known as *angioneurotic edema*, sometimes as *giant urticaria*. The most striking evidence of this affection is patchy swelling of the face. Very often the loose areolar tissue about the orbit is involved enough to close one or both eyes. The process of swelling comes on with striking rapidity. Some of the patches are fiery red, some white, some red and white. They are serpiginous in outline and they itch intolerably. Not only the face is involved; the dorsum of the hands and of the feet as well may be greatly swollen and the irregular giant urticarial wheals may appear at any place on the body. Sometimes they are so closely set that a whole limb or a large part of the trunk may appear edematous. In severe cases, especially when the seizures complicate tetany in an infant, edema of the larynx and of the lung may supervene. Such an edema in these situations may prove fatal. In any event their presence is sufficiently threatening to cause the greatest alarm.

There seems to be no doubt but that the appearance of *giant urticaria* is an anaphylactic phenomenon. It usually happens as the response of the tissues of a sensitized person to the advent of heterologous protein into the circulation. Such proteins find their way into the blood for the most part after ingestion, although sometimes they are injected by biting or stinging insects and at other times they may be absorbed directly from the mucous membranes of the respiratory tract. During infancy the com-

monest cause of such an anaphylactic outbreak is the ingestion of raw or insufficiently cooked egg white. Fortunately the reaction is not extreme in all sensitive infants. In a few it appears in the severe form together with edema about the glottis to form a most alarming clinical picture. Infants who show these severe manifestations are often either eczematous or asthmatic as well, sometimes both. Other foods may produce the same effect, but rarely to so great a degree as egg white does. Of the commoner edible articles, oatmeal is next often the source of such trouble. Rarely ingestion of bovine milk is met by this exaggerated anaphylactic response. More frequently it would seem that the protein of some unusual plants which the dairy cattle have been allowed to eat passes into the milk and produces an attack of giant urticaria in a susceptible infant who has received the milk.

The *treatment* of the condition resolves itself into measures taken to check the attack and to relieve the patient, and others instituted for the prevention of recurrences. Prompt relief of the itching, cutaneous swelling and of the edema of the larynx and lung usually follows the hypodermic injection of atropin and adrenalin. For a 25-pound baby, 1/300 grain of atropin dissolved in 10 minims of 1/3500 adrenalin solution may be given. As an adjuvant to the injection or without it in slighter cases, baths and local applications may be used. About the eyes iced compresses of boric acid solution are grateful. Baths should be medicated with phenol or cresol in the strength of 1 to 5000 or 6000. As a soothing local application phenol grains 10, zinc carbonate dram 1, in calamine lotion ounces 3, is comforting. The distress may be such that a single injection of codein 1/12 grain to a 25 pound infant may become necessary. Such an injection will naturally be withheld except under the stress of necessity. Calcium by mouth seems not only to aid in overcoming the symptoms of the acute attack but also to prevent future recurrence. Calcium lactate so often advised is much less satisfactory than calcium chloride. A palatable and efficient elixir of calcium chloride is purchasable. The dose for a 25-pound child is 1 teaspoonful; this contains 5 grains of calcium chloride. The drug may be given dissolved in water and be added to the food. When, as may happen, tetany underlies the appearance of the edema, milk should be entirely withdrawn from the diet. When it is possible to identify the protein to which the infant is sensitive an attempt should be made to immunize the child. Immunization to

egg white is readily accomplished by feeding minute doses of crystalline egg albumen. The initial dose is that just short of the amount which in dilution will give a skin reaction. The albumen is given daily in increasing increments, the size of which will depend on the child's response. Should it show even the slightest reaction, return should be made to a dose of the same size as the initial and a slower advance be made by using smaller augmentations. Much progress is being made in the preparation and use of other proteins for purposes of immunization and it is certain that success will often follow such therapeutic attempts.

Those who insist that breast feeding alone can bring a child to a satisfactory state of nutrition are apt to delay *weaning* and the institution of mixed feeding, with the result that infants reach the tenth or twelfth month so habituated to exclusive breast feeding that some of them are unwilling to accept food in any other form. For this reason, in spite of many published objections, the writers favor the plan which, under hygienic conditions only, provides a single daily bottle feeding, and later, at about the fourth or fifth month, a daily cereal meal as well. The freedom such a plan permits the mother, if the bottle is given during the day, or the unbroken sleep it provides for her if the feeding is at night, is of great advantage to her health, and tends to maintain or to increase her milk supply.

A gradual decrease in the number of breast feedings daily offered, is the better way to wean the child; for the amount of milk secreted gradually diminishes and finally ceases without discomfort. In general the breast feedings should be given up by the end of the tenth or eleventh month, except in those infants who have a proven idiosyncrasy for cow's milk.

In case it is desired to wean more rapidly, the child should be put in charge of someone other than the mother. If the child has passed its seventh month, there is some advantage in feeding it from a spoon, and so avoiding the use of the nursing bottle. Milk thickened with cereals and broths, to which sago, or pureed paste and finely divided vegetables are added can be made the basis of the meals.

The mother ought to restrict her fluid intake; she should secure two or three watery stools daily through the use of saline laxatives; and the breasts should be supported firmly by a tight brassiere. Whenever the breasts become painful, milk should be removed by manual expression; no more than enough just to relieve the feeling of tension should be taken out. Within a few days, all annoyance should cease.

CHAPTER VI

HEMORRHAGE

From the Mouth.—On the *appearance of blood from the mouth*, be it much or little, a search should be made of the buccal cavity for ulceration. Gingivitis, ulcerative stomatitis, whether due to B. fusiformis or streptococci, noma or syphilitic ulceration may be causative. The bleeding gums of scurvy are not uncommon. Submucous hemorrhages occur in the hemorrhagic diseases and in the severe exanthemata.

The *treatment* should be directed toward the cause. In cases of syphilis or scurvy, constitutional treatment produces immediate and happy results. Of local treatment, none is better than the application of a 50 per cent solution of trichloracetic acid, following the painting of the parts with a 5 per cent cocaine solution. In the treatment of gingivitis with spongy gums and small superficial ulcers, this drug (trichloracetic acid) is particularly useful. For its application, it is essential that a small carrier be used with cotton tightly wound and that the application be made directly to the gum margins and the ulcerated surfaces. Care should be taken that no excess acid runs down over the healthy tissues, as this drug is a very powerful caustic.

Nitrate of silver in 20 per cent solution, tincture of iron, or a 1 to 3500 adrenalin solution, may be used with advantage, but they are less effective than the trichloracetic acid.

In these conditions it is advantageous to spray the mouth and throat with an aqueous solution of one-tenth of one per cent zinc chloride or with one-fourth of one per cent phenol, in cinnamon water.

Hemorrhages following operation for the removal of tonsils and adenoids may be easily overlooked if the child is allowed to lie on its back. The patient is unable to expectorate, the blood is swallowed, and the hemorrhage may be revealed through a sudden hematemesis. Prophylaxis in such cases lies in the careful instruction of nurses who should be taught that frequent swallowing after tonsillectomy, is a danger signal and should be immediately reported to the surgeon. They should also be taught that with a child lying in the lateral position, the

blood escapes on the pillow and the presence of hemorrhage is thus revealed. Treatment consists of immediate stasis by clamp, pressure or ligature.

When blood is ejected from the stomach of an infant, during the first to the tenth day, even without the presence of bleeding elsewhere, the possibility that the child is suffering from *hemorrhagic disease of the newborn* must not be ignored. But neither should it be forgotten that blood vomited on the first day may have reached the stomach through a hemorrhage in the upper respiratory passages through trauma incident to birth.

From the Nose.—Bleeding from the nose does not occur with any great frequency during the first two years of life, and when it does happen, it is rarely excessive. Trauma and ulceration are the commonest causes. The former seldom needs more than a slight external pressure or a cotton or gauze plug in the anterior nares. Occasionally there is an erosion into a small vessel of the nasal septum. In such cases when the plug is removed the bleeding may recur. It may be checked by the destruction of the vessel by the actual cautery. On rare occasions, especially in hemophiliacs, it may be necessary to plug both the anterior and posterior nares. This is most readily accomplished by cutting an eye in a soft rubber catheter, inserting it into the anterior nares until it is thrown out through the mouth by pharyngeal spasm or brought out by hooking the index finger around it. Into the eye is threaded doubly a small cotton cord, to the ends of which is tied a piece of cotton or gauze sufficient in size to occlude the posterior nares. This is very gently pulled into place, the cords are separated and a like plug inserted into the anterior nares and the cords tied over it. In this manner both exits are closed, the nasal cavity soon fills, and the hemorrhage ceases. This procedure should be used only after all other hemostatic measures have failed and the plugs should be removed at the earliest possible moment as the closing of the pharyngeal ends of the Eustachian tubes may give rise to middle ear suppuration.

Epistaxis accompanying the *florid stage* of *syphilis* is rarely of sufficient severity to warrant intervention, other than that directed against the invading spirochete. In the *acute exanthematous diseases,* especially *diphtheria,* hemorrhage of the nose is sometimes annoying but it is rarely that a case does not respond to simple treatment.

From the Lungs.—Blood appearing at the lips from the lungs is practically unknown in infancy. The commonest cause of

hemoptysis, ulcerative tuberculosis, is a rarity at this age. There is sometimes a hemoptysis in pertussis due to the rupture of a vessel during a paroxysm. The lungs bleed in the pneumonias, but as the child is unable to expectorate, the blood does not appear at the mouth.

From the Stomach.—Hemorrhages may occur in *ulcer of the stomach, scurvy, purpura, leucemia* and in the other *hemorrhagic* disorders. It often follows *trauma* to the gastric mucosa incident to stomach lavage. It should be borne in mind that blood seen in the vomitus may have been swallowed and a search therefore should be made for a source of bleeding in the mouth, nose or throat. The treatment of these conditions must be directed toward the cause.

From the Intestine.—Intestinal hemorrhages are made evident by the presence of blood in the stools. It is obvious that unchanged blood must arise from the lower bowel and that dark tarry masses proceed from the higher levels. Occurring in the newborn, such an evacuation, either alone or accompanied by bleeding from other sources, is evidence either of *idiopathic hemorrhagic disease (hemophilia), sepsis or syphilis.*

The preferred treatment is simple and effective and consists of the intramuscular injection of whole blood which has been taken from a healthy donor, preferably the father of the child. For the technic see Methods.

Later in infancy one of the most important sources of bleeding is the infolded bowel of an *intussusception.* This accident is rare before the fifth month and unusual later than the fifteenth month of infancy. The hemorrhage is characteristically intermixed with mucus and appears in an evacuation mass that looks like currant jelly. The treatment is by hydrostatic reposition or by surgical intervention. In *volvulus* the character of the hemorrhage is so similar to that of intussusception, that the bleeding renders no aid in differentiating the two conditions.

Ulcerative tuberculosis of the intestine, while a potential source of bleeding is an uncommon thing in the United States. When it does occur, it is usually one manifestation of a widespread tuberculosis. The same statements may be made of *typhoid fever and influenza. Scurvy,* on the other hand, is not infrequent and a moderate degree of melena may characterize it. At the age of infancy with which we are dealing, hemorrhages are most commonly seen as complication of infectious diarrheas, particularly those in which the dysentery group of bacteria plays an etiologic rôle. In dealing with these hemorrhages, the only effective measures are

those which may be taken in conjunction with the treatment of the diarrheas. These are discussed in another chapter. The cure of the melena of scurvy will follow constitutional treatment.

The presence of bright red blood stains or clots on the diapers, may indicate the presence of *fissure in ano, polypi, hemorrhoids, proctitis or ulceration*. The hemorrhage which accompanies fissure in ano is usually trivial. *Polypi* may grow from the mucous membrane as high as the sigmoid. They are friable, easily injured and may be the source of profuse hemorrhage. As a rule, the only other symptom that accompanies their presence is the straining which indicates the child's attempt to empty the rectum. This symptom may antedate by a long time the presence of blood in the stools. The treatment is excision of the growth. Infants are so seldom subject to *hemorrhoids* that these dilatations may safely be ignored as a source of bleeding. When there is any bleeding from the pile-bearing area, thrombosis usually occurs, obviating the necessity of treatment.

Isaac Abt has called attention to the fact that *calomel* may cause mild hemorrhages from the bowel in susceptible subjects. The use of *syrup of hydriodic acid* is sometimes followed by slightly blood-stained evacuations from the bowel.

From the Genitals and Urinary Tract.—Blood in the urine of the newborn may be evidence of *trauma or of hemorrhagic disease*. During the first and second years, *scurvy* is a fertile source of hematuria. In the latter period, *concretions* usually of uric acid, may traumatize the urinary passages with the same result. *Bacteriemias and toxemias* are agents that occasionally produce a hemorrhagic nephritis. A proportion of children suffering from *colon bacillus cystitis* show macroscopic blood in their urines although in but few instances is there profuse bleeding. *Tumors of the kidney*, and with less frequency of the bladder may be responsible for the appearance of blood. Fortunately the complicating *nephritis of scarlet fever* is not often encountered during the first two years of life. However, when it does occur, the outlook is grave. The bleeding is purely glomerular. Rest is, par excellence, the treatment of an inflamed tissue, and the only means we have of resting the glomeruli is by restricting the intake of fluid and in this way diminishing the work of these kidney filters. The detail of this treatment is dealt with in the chapter on Genitourinary Diseases.

On occasions, considerable *hemorrhage may occur from the vagina of a newborn girl*. Many women believe in the menstruation of

the newborn. This bleeding is either traumatic or it is evidence of hemorrhagic disease. Retraction of the prepuce in boy infants may be followed by a severe hemorrhage which ordinarily can be controlled by pressure, but if the frenum be torn, a ligature may be necessary before the bleeding is controlled.

From the Skin.—Hemorrhages into the skin, scalp and subcutaneous tissues may be traumatic in origin or they may be evidences of constitutional disorders. *Cephalhematoma,* in which blood accumulates in the loose areolar tissues of the scalp during the passage of the head through the birth canal, is the commonest of the massive hemorrhages due to trauma. The only treatment is "masterly inactivity." Interference is to be deprecated.

Hemorrhage into the sternomastoid muscle is often accompanied by a rupture of some of the fibers of the muscle and is followed by a shortening which gives rise to wry-neck. Often when the bleeding is slight there is no need for treatment, but ordinarily heat and massage will hasten absorption and tend to restore the pliancy of the muscle, and in this way the torticollis may be forestalled. Other hemorrhages due to injury are unimportant.

Blood extravasation during the first week of life without traumatic etiology is pathognomonic of *morbus maculosus neonatorum,* or is of the hemophiliac or of the purpuric type. The hemorrhages may be extensive or slight, but almost always they are accompanied by evidence of bleeding into the internal organs. It is important that the physician should promptly inject human blood intramuscularly or human or animal serum into the circulation. The use of gelatin and calcium is obsolete.

Lucas, of the University of California, in investigating blood conditions in the newborn, especially with relation to coagulation time and the elements concerned in its prolongation or retardation, has found that during the first few days of life, blood coagulation time is increased in normal children as well as in those with a tendency to bleed; and that this increased coagulation time seems to be due to a deficiency in prothrombin. The insufficiency of the data in hand makes Lucas unwilling to accept this as the only factor in the changed coagulation time. The problem is made more difficult by the presence of bile, in quite large quantities, in the circulating blood of many newborns. This apparent reduction in prothrombin is of great importance because of a similar finding in hemophilia, and in those hemor-

rhagic diseases of the newborn, in which sepsis is not an etiologic factor.

As a complication of hemorrhagic disease of the newborn, sometimes even its only symptom, *umbilical hemorrhage* is important. The bleeding varies in intensity from slight oozing to a copius spontaneous hemorrhage. Local applications of kephalin or thromboplastin are useful especially in the oozing cases. Failing these, a piece of freshly cut muscle from a rabbit or guinea pig may prove of value because of its thromboplastin content. These local treatments can be only palliative, and for a cure blood injections must be depended upon. If the case falls into the purpuric category, *sepsis* and *syphilis* must be sought for and treated if present. The umbilicus must be kept under strict surveillance because severe infections may occur here with but few visible signs. All dead and sloughing tissue must be carefully removed; compresses of glycerin and alcohol should be applied to the infected area and frequently renewed.

The purpuras of later infancy are identical in their manifestations with those just described. They are, however, more often symptomatic and may occur in the course of a *focal infection* especially *one of tonsillar origin. Miliary tuberculosis* and any of the other general infections, especially the *malignant forms of diphtheria, scarlet fever and typhus* may also be causative. *Leucemia* and the *primary anemias* on very rare occasions may be etiologic. Again blood injection is the remedy of choice for checking the hemorrhages. It can only be adjuvant, however, to measures directed against the underlying causes.

From the eighth to the eighteenth month of infantile life, the most frequent cause of hemorrhages into the skin is *scurvy*. While bleeding is not the most striking symptom, its presence renders diagnosis certain. Tenderness, pseudoparalysis, spongy and bleeding gums and purpuric spots on the skin form a combination that cannot be mistaken.

In the course of a scurvy, bleeding may take place into the orbit to such a degree that the eyeball is pushed forward. A unilateral proptosis results. On occasion, bleeding into the gums and skin may fail, and then the proptosis alone will reveal the scurvy.

Recovery from this disease rapidly follows the administration of the antiscorbutics. The treatment of this disorder is dealt with in the chapter of Nutrition.

Into the Viscera—

Bleeding into the eye may be accompanied by visible post-choroidal hemorrhage with a detachment of the retina. This unhappy accident may result from natal trauma or occur as one event in the course of hemorrhagic disease of the newborn.

As a result of trauma or of one of the hemorrhagic diatheses, bleeding into any of the *abdominal viscera* may occur. There are no visible evidences of hemorrhage under such conditions and the symptoms will be referable to changes in the function of the organ damaged by the bleeding.

Treatment consists of the injection of whole blood or blood transfusion. (See chapter on Methods.)

Autopsy shows that about half of the infants who die within a week after their birth have suffered *intracranial hemorrhage.* Sometimes the damage is to the veins entering the longitudinal sinus, when blood is poured out over the cerebral cortex. In other circumstances, it is the rupture of the transverse sinus or of vessels broken when the tentorium is torn, that permits blood to gather and clot about the base of the brain. Again, the hemorrhage may be intraventricular, arising because the veins of the choroid plexus have given away. Warwick and Rodda have brought evidence of an increased bleeding and clotting time in the blood of many infants, the victims of such hemorrhages. They believe that their findings indicate that many such suffer from hemorrhagic disease of the newborn, and that the treatment should include immediate intramuscular injection of whole blood, or a blood transfusion.

The earliest clinical evidence of intracranial hemorrhage usually is somnolence; this may not supervene for a day or two after birth. Very early twitching of one or more muscle groups comes on, to be followed by spasticity—usually of the lower extremities—in which adductor spasm is noteworthy. Increasing intracranial pressure causes the fontanelle to bulge and brings about disturbances of the respiratory rhythm, at first slight, later in the nature of Biot grouped breathing—a phenomenon sometimes mistakenly called Cheyne-Stokes breathing. The pulse is also apt to slow and to become irregular. Eye signs—nystagmus, strabismus or fixation and irregularity of the pupillary reaction—appear early, but they may be absent until after the pressure becomes extreme. Convulsions always appear early; they come on when the bleeding is over the motor area, later when it is at the base or within the ventricles.

When a newborn infant lapses into somnolence and persistent cyanosis, and shows a tendency to breathe irregularly, and has a fontanelle which seems fuller than normal, lumbar puncture is indicated. If bloody fluid flows from the needle hemorrhage is probable. But the ease with which the anterior plexus may be torn must be remembered, for blood flowing from this source will obscure the diagnosis. Under these circumstances 10 to 15 c.c. of fluid should be removed daily until such times as the flow is unclouded by blood.

If the ophthalmoscope shows edema of the disc in an infant with bulging fontanelle, even if the spinal fluid is clear, the advisability of performing a subtemporal decompression must be promptly considered, especially if the spinal fluid flows under greatly increased pressure.

CHAPTER VII

PAIN AND TENDERNESS

Despite the incomplete organization of its nervous system, pain may afflict the infant from the moment of birth. Tactile and visceral sensation are present at the onset of postnatal life. In its early days, the infant has little discriminatory power, with the result that slight pains elicit as great protest as severe ones. The little baby has no means of expressing his pain other than by crying and screaming. As the weeks pass, discrimination develops, minor pains are sensed and give rise to motor phenomena such as wriggling, grimacing, whining and whimpering. Still later as the child passes toward the end of the first year, motor coordinations are such that the movements of the hands will often indicate the site of the pain if it is localized. The infant's tendency is to carry the hand to the painful part whether it be abdomen, ear or epiphysis. Even very young infants express agony by a sudden intense scream. The poignancy of pain depends to a very large degree upon the pressure upon the nerve endings of the affected organ. For this reason, most intense distress follows inflammation and effusion in sites such as the middle ear where the periosteum is firmly attached.

The **ear** is one of the most frequent sources of pain in infancy. *Inflammation of the middle ear* with bulging drums often occurs and almost always intense pain accompanies such an affliction. Such distress is characteristically intermittent and causes the infant to scream violently with a piercing, shrill shriek, repeated two or three times. This is followed by a few minutes of whining or sobbing, then by a period of normal behavior which may last from 10 minutes to perhaps several hours before the pain again forces the child to protest in the same way. Under these circumstances, an examination of the ears will reveal reddened or bulging ear drums. Most often fever will be a feature. Involvement of the throat may be found in reddened, swollen fauces. Often the cervical glands, anterior and posterior, as well as the posterior auricular glands will be swollen and tender.

Application of dry heat externally by hastening suppuration and diminishing sensibility brings relief. Such relief will be more prompt and thorough if a few drops of a 2 per cent solution of

phenol in glycerin are instilled into the external auditory canal. The anesthetic power of the phenol, together with the hygroscopic and softening properties of the glycerin, combine to diminish the pain often with remarkable celerity. Some aurists prefer to use 2 per cent cocaine in a solution of 1 part of adrenalin to 10,000 parts of water.

After all, such treatment is but temporizing and confronted with a sick and unhappy child whose ear drums are swollen, we should not hesitate to incise the membrane at once. Merely to puncture the drum is not enough. Such a simple stab wound may quickly close and all the symptoms may recur. The procedure is so simple that the aid of an aurist is not always imperative. The Von Grafe cataract knife is the best cutting instrument to use. A good artificial light is essential. This may be thrown on the drum by an ordinary head mirror or the membrane may be inspected by aid of one of the excellent illuminating specula available. Some such outfit, including the knife, should form part of the equipment of the physician's handbag and as well, find a place in his office outfit. It is rarely necessary to give an anesthetic to accomplish a paracentesis tympani.

It is important to have the child properly held; for examinations or manipulations about the throat, the child should be enveloped in a blanket that will cover it from shoulders to feet. This should be rolled in such a way that the arms and legs are prevented from moving. The little one is then brought into an erect position and firmly held so by an attendant who clasps the patient close to her bosom. (See Methods.) The speculum is then inserted, the drum well illuminated, the Von Grafe knife plunged through the point of greatest bulging and the blade carried upward and backward at an angle of 30° or thereabouts. Pus or serum or a few drops of blood may follow the knife as it is withdrawn. In any event, relief of pain will be prompt and certain. It will be necessary, however, for some days to douche the ear at intervals. For this purpose there is nothing better or more readily obtained than hot normal salt solution. One-third teaspoonful of common salt dissolved in a tumbler of boiled water is easily prepared. The fluid is forced past the meatus by gentle pressure from a soft rubber aural bulb syringe. Three or four bulbfuls will be needed. Care should be taken to have the head bent to the side of the suppurating ear in order to facilitate the emptying of the canal. This is further aided by tucking absorbent cotton into the meatal opening.

Pain in the throat may occur alone or in combination with earache. Under the latter circumstances, the symptoms of earache will predominate and the treatment of the ear condition will be the urgent need. Painful throat may cause a baby to fret, especially at meal times, but the pain is rarely severe enough to cause the child to cry continuously. Swallowing increases the pain, often to such a degree that food is refused or poorly taken with much protest. In certain throat infections, the fauces and tonsils are involved but the discomfort is produced or the pain aggravated by a concomitant *adenitis* or *myositis of the neck muscles* that produces guarding and stiff neck; or if the sternomastoid muscle is involved, wry neck. At times the involvement of the neck muscles may be so great that there is difficulty in differentiating the condition from the rigidity of a meningitis.

The *treatment* is local and constitutional. Before undertaking the local treatment of faucial inflammation in children, it is essential to restrain the child by rolling him in a blanket that reaches from the shoulders to below the feet and which is firmly tucked around him. The child is held in the erect position, the head is firmly supported and the jaws are opened by a tongue blade. The jaws of a young baby have great power and the ordinary wooden tongue depressor is often entirely inadequate to force apart the jaws in order to obtain a good view of the fauces. (A proper metal blade or the handle of a tablespoon prove much more effective instruments.) Once the mouth is well open, a good illumination is essential and then after a thorough inspection, if need be, a sterile swab to obtain cultures should be employed. One culture should be made on glucose agar and one on blood serum. The inflamed fauces are then to be painted with a 10 per cent or 15 per cent solution of silver nitrate freshly prepared. Twenty per cent argyrol is less painful and sometimes is effective. It gives a better result, if it is used subsequently to 1 or 2 applications of silver nitrate. In using the latter drug, great care should be exercised that the swab does not contain an excess of the solution, which may run down and irritate the glottis. In young children, spasm of the larynx may follow careless faucial applications of irritating substances. Iodine solutions are also valuable as applications to inflamed tonsils. Sprays are available for use in late infancy. They also are more effective if they are used after a preliminary painting with silver nitrate. One-tenth of 1 per cent of zinc chloride and ¼ of 1 per cent phenol in cinnamon water makes a pleasant solution that may be used in either the nose or throat.

When myositis is pronounced, hot packs to the neck renewed every 2 or 3 hours are comforting. At the time of changing, gentle massage of the neck muscles is a great help in that it eases the child and hastens absorption. For mild degrees of adenitis and slight myositis, massage twice or thrice daily with an ointment of 10 per cent methyl salicylate in lard is effective.

Even as early as the day of birth, **adenoids** may be sources of discomfort. Often during the first month, especially after an attack or two of snuffles, the adenoid tissue enlarges so that the narrow nasopharynx is obstructed either intermittently or continuously. Such a blocking is a frequent cause of distress and crying in little babies. It is also one reason for the opisthotonos of young infants who assume a position lying on the side with retracted head to favor the entry of air into the lungs. The presence of an adenoid may be enough to occlude the pharyngeal end of one or both of the eustachian tubes and at times to produce a negative pressure within the middle ear, with the result that the unbalanced atmospheric pressure may cause earache. When adenoids obstruct the nostrils, nursing is interfered with, sleep is disturbed and much mild distress is engendered. Sometimes even when the adenoid mass is small, intermittent obstruction is brought about by a congestion. Under those circumstances, instillation 2 or 3 times a day of an oily mixture of 10 per cent gray oil, 25 per cent adrenalin ointment and 65 per cent liquid petrolatum or almond oil is very effective in relieving the obstruction, diminishing the distress and eradicating pain. At the same time, 4 or 5 doses of atropine in sugar water may be given for its effect in diminishing secretion and drying the mucous membranes. The dose should be 1/1500 grain at 2 hour intervals for a 15 pound baby, until dryness of the mouth and flushing of the face occurs. It is useless to give atropine unless the physiologic maximum is reached, and this reached, the drug should be discontinued at once.

Headache is often an accompaniment of infections, especially of those that afflict the throat. Crying and rotary movement of the head are suggestive of headache in infants. Sometimes older infants will bore the head into the pillow, and toward the end of the first year of life, the hands will be carried to the head. In adults, increase in intracranial pressure will cause headache but in the case of the young baby the open fontanels and easily separated sutures allow relief of the pressure and not until attention has been called by the bulging of the fontanel or the wide

separation of the sutures with an increase in cranial dimensions, does obvious headache become a prominent symptom of such affections as hydrocephalus, meningitis and brain tumor.

The *treatment* of headache resolves itself into measures that mitigate the fever and toxemias arising from infection, although small doses of pyramidon, (½ to 1 grain for 2 or 3 doses for a 15-pound child), may be justified if the pain is excessive. Often the addition of an alkali, (potassium citrate, 8 grains or sodium bicarbonate, 4 grains) to the food will act as an effective adjuvant to measures directed against the local infection.

The anal and rectal regions of the infant should not be overlooked, because no other parts of the body except the upper respiratory and ear tract are so frequently the sources of distress and crying. **Proctitis, ulcerations** and particularly **fissure in ano with spasm of the sphincter,** are to be searched for when the child evidences much discomfort. A great deal of the so-called "colic" of babies has its origin in the terminal bowel. Fissures need not be large to be exquisitely painful nor need ulcerations be visible to cause great distress. An anal sphincter that is hard and hypertrophied is sufficient evidence of some pathologic state in the anal canal or lower rectum. Coupled with pain on passage of gas or feces, this evidence becomes incontrovertible. Such a child often gives evidence of distress that is interpreted as arising from intestinal colic. On the ingestion of food, the intestine is aroused, the peristaltic wave rolls on until it reaches the tender termination of the gut. Immediately pain is felt, the sphincter goes into spasm, still further accentuating the distress, and the child draws up its legs and screams repeatedly.

If slight, such a state of affairs can be remedied by local applications of 10 per cent belladonna, and 15 per cent ichthyol in cold cream or lanolin. A simple and painless way to apply the ointment is to use a large, soft, rubber, male catheter (No. 16 to 20, English), and to have its tip well smeared and the eye filled with the ointment and to gently insert it into the rectum. When a catheter of this size is used, it serves to dilate the sphincter and to apply the ointment which is both antispasmodic and soothing. The eye is opposed to the ulceration or fissure, the catheter is then withdrawn with one hand while the thumb and finger of the other milk out the ointment from the eye as the tube is drawn out between them.

In severe or neglected cases with great hypertrophy of the sphincter, manual dilation must be used, preferably after a slight

anesthetic; or the fissure may be painted with 2 per cent cocaine solution followed by 20 per cent silver nitrate. Only rarely will it be necessary to incise a fissure or to sever a sphincter as often has to be done in later life.

There is no more painful affliction at any age than fissure-in-ano and in the earlier stages of treatment it may be necessary to give narcotics to relieve pain and obtain sleep for the child. One-twentieth grain of codeine in water by mouth or $\frac{1}{30}$ by hypodermic injection for a 15-pound infant, may be given and this may be repeated once after 6 hours.

Abdominal distress or **colic** is usually classed as the commonest cause of the pain and discomfort that lead to a baby's unhappiness and crying. This may have been so before the technic of feeding, both breast and artificial, had attained its present level of efficiency. Today among well-fed, well-cared-for infants, discomfort from food cannot be said to be common. Much unhappiness is imposed on babies because of the attitude that every mother who has an ample breast secretion has therefore a milk that will agree with her child. No greater fallacy could be promulgated. A good human breast milk that agrees is undoubtedly the finest food for the human infant, but a wisely devised, properly prepared, artificial feeding is vastly better than a poor breast milk. One of the commonest causes of distress in breast-fed infants is an insistance that the child should nurse exclusively from an inadequately secreting breast or on breast milk which after a fair trial has been demonstrated not to agree with the child. Of course an attempt covering at least 3 weeks must be made to preserve breast feeding in whole or in part. If, as often happens, the sugar is too high, dilution by feeding water just before nursing will often relieve the distress. If the secretion is overweighted with fat, an increase in the nursing interval usually is effective, and if this practice fails, the giving of a teaspoonful of lime water with a little water may be helpful. It should not be forgotten that supplemental feeding can always be employed and that some maternal feedings each day are usually desirable.

The child who is overfed suffers from hyperchlorhydria which he translates as hunger. It is rare that the infant who seems famished, who lies with a dry, red, open mouth, its head turning from side to side eagerly seizing everything that is offered and ravenously attacking the bottle when it is given, is in truth

hungry. In reality, he is suffering from "heart burn," *acute indigestion,* from too much food or from a feeding poorly balanced. An increase of his ration is followed by a corresponding increase of the intensity of the symptoms. A few hours limitation to water and a dilution of the food ordinarily will restore tranquillity and comfort in short order.

These symptoms may appear in the bottle-fed as well as in the breast-fed. Here the treatment consists of modifying the artificial food. A fond mother or a nurse, anxious to produce a rapid gain in weight, may have unduly increased the fat by using cream or top milk, or she may have run up the sugar beyond the amount ordered because someone else's baby has been doing well on such amounts. The type of vomiting will often determine which food element is out of balance, whether fat or sugar. A preliminary 12-hour withdrawal of food will be the first step toward getting the child comfortable. If fat has been at fault, the new feeding will begin with a fat-free formula, made from skimmed-milk, with a fairly high sugar (1¼ to 1½ ounces to a 20 ounce mixture). After 3 days, fat may be begun (½ ounce of the top 5 ounces of a quart of milk on which the cream is well risen may be added to a 20 ounce mixture). After 2 days, another ½ ounce may be added then the top milk increased carefully every few days until the mixture contains 2 to 2½ ounces of the top 5 in 20 ounces. More fat than this is almost certain to bring about a recurrence of the distress.

If carbohydrate is in excess, relief can be obtained by reducing the sugar content and by using a dextrin such as flour ball or baked flour instead of the sugar. We may begin with ⅓ ounce in a 20 ounce mixture and increase slowly from day to day until 1 ounce has been added to each 20 ounces of the formula. Often such babies do better on the type of formula advised by Brady of Kansas City which he has called the polycarbohydrate formula. Such a feeding might call for ⅓ ounce of malt-dextrin, ⅓ ounce of dextrin or starch and ⅓ ounce saccharose. The principles of these feedings are discussed in the chapter on Nutrition and the preparation of the formulas is detailed in the section on Formulas.

Underfed babies are also uncomfortable but their hunger rarely causes them to exhibit such a degree of distress as one sees in the case of the overfed. Underfeeding is more often made apparent by loss of weight, apathy and a certain doleful expression of the patient. The incessant wolf-like search for food is not

found in these little ones except in those cases in which indigestion is also a factor in producing pain.

Prompt alleviation of such discomfort follows the establishment of an appropriate complementary feeding in the breast-fed, or an increase in the quantity and quality of the food prepared for the artificially-nurtured. It is an interesting commentary on the teaching influence of the medical profession that today, underfed babies predominate among those who are brought to the physician for nutritional disturbances. A few years ago in this group of patients almost all were overfed but under the insistent preachment against overfeeding there has grown up a generation of mothers, nurses and young doctors who are so fearful of this error that many of them swing to an opposite extreme.

Colic, that is actual spasm of the intestine following irritation elsewhere than at its termination, may follow certain types of unsuitable feeding in young babies during the first 3 or 4 months of life. It may occur even with almost perfect dietetic regulation, because in certain infants at this age, the nervous control of the intestine and of intestinal movements is ill developed.

In our opinion, contrary to the teaching that casein never causes indigestion or distress, it is probable that the large casein curds which sometimes enter the intestine when certain milks, unboiled or otherwise unmodified, are fed, become sources of distress, for they may act in the gut as foreign bodies. As a result, irritation is induced and spasm and pain occur. Simple boiling of the milk is a fairly effective way of overcoming this painful condition. If it be boiled with starch, which acts as a colloid, and dextrin as in the preparation of malt-soup, the results are more certain. The addition of citrate of soda to the raw milk or after boiling is also useful, but rarely necessary.

Flatulence is another cause of spasm in the intestine with pain and distress. This excessive gas accumulation may result from several causes. Gas accumulation is not always gas production. Air is often swallowed by the nursing child, particularly if it be bottle-fed, sometimes to its great discomfort. The erect position during and after the meal is the remedy.

Flatulence may come from an inefficient circulation. This result is usual in the congenitally enfeebled and in those who have a congenital defect of the heart such that blood pressure and circulatory volume are interfered with. In these cases, respiration is inefficient and carbon dioxide passes

over into the intestine. The *treatment* here must be directed to maintaining the balance of circulation and to encouraging efficient heart action. Warmth is an essential aid in the treatment of these patients. It may be applied through the agency of electric pads or blankets. In severe cases, the use of the mustard bath is of great value, both for the warmth it supplies and for its stimulating effect on the circulation. When less severe, hot applications to the belly may suffice, and occasionally, a warm saline enema with tincture of asafetida added is helpful.

In order to prevent crowding of the diaphragm and embarrassment of the weakly acting heart, small meals should be given such babies, and being small they should be less diluted than the food offered normal babies of the same age or size. Being concentrated, it is well partially to digest such meals by the use of peptonizing powders; either the hot or cold process may be used. (See Formulas.) Generous additions of the sugars up to 8 per cent are indicated whenever the circulatory apparatus is acting weakly.

In the treatment of this condition the value of drugs is questionable. The addition of a teaspoonful of cinnamon water to each feeding is recommended by some English writers; it can do no harm and sometimes it seems to act with good effect. The injection of 3 to 5 minims of 10% camphor in sterile olive oil is very valuable in some cases. Infusions of camomile or caraway seed are household remedies, both harmless, and they are apparently comforting to the nurses and mothers of the unhappy infants.

A single purge is of value when colic arises from foods in which bacterial decomposition has taken place. It should be used in the colics of circulatory origin with great caution.

By far the commonest source of distention that leads to pain, discomfort and colic is an implantation of gas forming bacilli among the flora of the infant's intestine. B. Welchii is the commonest offender. The mode of its growth and the manner of its life have been dealt with in the chapter on Diarrhea and there the method of treatment looking to its elimination is given. Sufficient to say in epitome, that the treatment is first to give the patient 1 to 3 drams of castor oil and then to initiate a 24-hour period of egg albumen water feeding. (See Formulas.) Following this for 24 hours protein-milk, without carbohydrate addition, is given. For the next few days carbohydrate (corn syrup) in daily increments of ½ to 1 per cent, until 5 per cent concentration is reached, is added to the protein-milk. Upon withdrawing protein-milk, skimmed-milk

mixtures should be used, and the cream ration increased by adding daily ⅓ ounces of the top 5 ounces of a quart of milk (top 5 ounces has 20 per cent of cream), until this added top-milk ration reaches 1⅓ ounces in a 20 ounce mixture. The milk should be boiled and no raw milk should be given such a child for several weeks.

In dealing with a case of colic, especially if it be of long standing where the great distress and sleeplessness of the child has upset the equinimity of the household, it may be necessary to give opiates to the infant in order that it may sleep. This we may have to do in order to calm the anxieties of the mother, to obtain for her a full sleep and to tranquilize the environment for the good of the patient. By hypodermic injection, or by mouth, we may make use of codein. If subcutaneously, a fair single dose for a 15-pound infant is $\frac{1}{30}$ grain. If by mouth $\frac{1}{20}$ grain may be used, which may be combined with ½ minim of spirits of chloroform and ½ teaspoonful each of simple elixir, and of water.

In any case, before intestinal colic is diagnosed, it will be wise, carefully to canvass the possibility of fissure-in-ano, ulceration of the rectum or of a gastric indigestion of the hyperchlorhydria type.

Constipation often causes discomfort and distress, which is much aggravated when there is tenderness at the anal orifice. Even when this region is perfectly normal, the presence of a hard fecal mass may originate discomfort and crying that ceases only after an evacuation. Under such circumstances, an oil enema is indicated or if this fails to produce prompt emptying of the bowel, one of soap-suds may be used. Repeated enemata, however, must be avoided; constipation should be prevented by appropriate dietetic and general measures.

Pain and tenderness giving rise to much outcry often arises in infancy from **disturbances of the bones, joints** and **muscles.** In the first weeks of life, especially about the third or fourth day, it is common to encounter a baby who is dehydrated from vomiting, or diarrhea, or perhaps simply from starvation and water deprivation. The more extreme of such patients develop **pseudotetanus** in which the loss of water leaves the muscles hard and contracted. The child is so rigid that it can be picked up by the legs without any flexion following at the joints. The thighs are drawn upon the body, the head retracted and all flexibility has been lost. Spontaneous pain as well as tenderness is very great. Such infants cry incessantly and sleep but little.

The pain and tenderness follows the loss of water with the resulting changes in the muscles and it can be relieved only by restoration of the water balance, a result that can be brought about promptly and completely by the intraperitoneal or subcutaneous injection of water. (See chapter on Methods.) While dehydration is common in the first month of life, the fact that such a condition can be the cause of pain at any time during babyhood is often overlooked.

Rheumatism and painful arthritis are so rare in the first two years of life that it is almost the truth to say that they never occur. On the other hand, in the early stages of an **acute epiphysitis,** pain is a common symptom which will be announced by intractable crying, sometimes by screaming. In the very young, the upper epiphysis of the femur is the one most often affected, although the upper epiphysis of the humerus and the lower end of the radius are often involved. The treatment is by surgical incision, a procedure that must not be neglected or delayed, for these localized infections may become rapidly generalized. In this connection the possibility of the presence of scurvy must always be kept in mind. The subperiosteal hemorrhages of scorbutus sometimes resemble the tumefaction of pyogenic infection and unwarrantable surgical interference under such circumstances is not unknown.

In early life, **syphilitic periostitis with epiphysitis** is a rare cause of excruciating pain. The most common site for this inflammatory condition is the upper epiphysis of the humerus. Disability of the arm, and pain, together with a characteristic posture, aid in the diagnosis. Relief of pain promptly follows specific treatment either with mercurial inunctions, or by arsphenamine injections.

Toward the end of the first year and in the early part of the second, bone and muscle tenderness caused by **scurvy** is encountered with a fair degree of frequency. When the scurvy is frank and well developed, no trouble will be experienced in making a diagnosis. The pseudopalsy, and subperiosteal hemorrhages, together with the marked tenderness and pain, will be enough; but in the subacute or "fruste" types where pain and tenderness are almost the only symptoms, the diagnosis is more difficult. It is probable that a fair percentage of uncomfortable babies that cry when they are handled are in reality scorbutics of the subacute type.

Relief of the pain, so prompt that it seems almost miraculous, follows the inclusion in the diet of uncooked milk, orange juice

and potato cream. These additions will in a few days transform the tortured scorbutic infant into a smiling, happy baby.

During the early months, certain babies are subject to attacks of screaming, sometimes intractable, which continue for hours and are of frequent recurrence. At other times, the crying is moderate and fitful, recurring infrequently. These attacks are nearly always followed by the appearance of **uric acid** stains on the napkins. In the urine of those subject to the more severe attacks, tiny crystals of uric acid often may be found. In the later months, similar distress follows a concentration of urine that may occur from any cause which results in dehydration. Part of the distress, suffered by the dried-out infant, is undoubtedly due to such a urinary concentration.

The *treatment* consists in supplying water freely. However, the uric acid excretion of the early months is little affected by water ingestion. The addition of 1 minim of dilute aromatic hydrochloric acid to each ounce of water given is supposed by some to be helpful; but it is probable that no remedy is really effective in this condition. Mustard baths and the application of heat externally to the loins and abdomen is the most useful procedure at our command.

Very rarely infants in the second year develop **renal calculi** and when patients of this age are suffering from pain that manifestly arises in the genitourinary tract, this fact must not be lost sight of.

In boy babies, a **tight prepuce** may be the cause of great distress. This is not likely to occur, however, while there is room for even a narrow urinary stream to pass. In such an event, circumcision will be in order for constriction of the foreskin will be so great that forcible dilatation will be impracticable.

Pyelitis and **cystitis** are very common affections of girl babies. They occur oftenest between the sixth and sixteenth month and are rare during the first few months. Our youngest case was twelve days old. Most often there is little pain accompanying these conditions, but, on the other hand, some of the most intense pains in babyhood are encountered in these diseases, especially if the kidney be swollen or if there be ulceration of the urethra or ureter or a narrowing of either following ulceration.

Relief will follow a removal of the cause by appropriate treatment, local and internal, but the use of heat externally to abdomen and loins will be found an excellent adjuvant. In the

severest cases, heat is best applied through the medium of a mustard bath. At the outset of the extraordinarily severe pain that may accompany any of the painful urinary disorders, the physician may have to resort to the use of opiates. In such an event, a full dose is necessary and morphine sulphate is more effective than codeine. To a 15 pound baby in great agony, after a mustard pack or bath has been given without relief, one may use $\frac{1}{40}$ grain morphine sulphate with $\frac{1}{1000}$ of atropin, given hypodermically. The same treatment is applicable, if the pain and distress of renal calculus should render treatment necessary while the patient awaits surgical intervention. If this is not enough ½ as much may be given in 2 to 4 hours. It is to be remembered that in young infants, there is often a delayed effect from opiates and no surprise need to be expressed if an infant is somewhat stuporous for 24 hours following such a dose. There is no danger unless the child shows respiratory irregularity and a pin-point pupil. Should such untoward symptoms appear, external heat, atropin and artificial respiration will restore the infant. (For further data on treatment, see chapter on Genitourinary Diseases.)

The pain of uncomplicated **pleurisy** is distressing and is evidenced by a sharp cry on the part of the child, whose breathing is usually rapid and shallow. When, as is often the case, the pleurisy complicates a **bronchopneumonia or a lobar pneumonia** in its early stages, the cough of the disease aggravates the pain caused by the pleurisy. Under these circumstances, the use of opiates is indicated, and of these, the writers prefer codeine hypodermically ($\frac{1}{30}$ grain for a 15 pound baby), not to be repeated for at least 6 or 8 hours. Local application of light mustard compresses or plasters to the chest is sometimes helpful. Measures directed toward allaying cough, such as the inhalation of steam, may contribute somewhat to the relief of the pain. Strapping of the chest which is effective in older children and adults, is not to be advised in the case of infants.

Excoriation about the buttocks and **intertrigo** are among the commonest causes of crying in infancy and the agony that comes from the itching of *eczema* is almost intolerable.

Cleanliness, insistence on proper changing of diapers and the assurance that these are not washed with irritating, coarse soaps and are properly rinsed, will go far toward preventing irritation of the buttocks. After the toilet, lanolin should be used liberally especially in the groins and if a little stearate of

zinc is dusted over it, the protective effect will be enhanced. Once excoriation and denudation of epithelium has set in, the open treatment is necessary. The diapers are left off, a pad is prepared with two diapers, or clean cloths enclosing a layer of absorbent cotton, and the child is laid on its stomach on a pillow with the pad arranged to catch the discharges. The excoriated areas are left exposed to the air and to diffused (not direct) sunlight. The bed clothing must be arranged to insure warmth unless the room can be kept at a temperature of 70° or thereabouts. Such management, if properly carried out will cure the most excoriated buttocks in the course of a few days. (See Methods.) If there is much oozing, a few hours application of a lotion made with equal parts of milk of magnesia, black wash and rose water with a few drops of phenol added, will facilitate the healing.

Urticaria is a frequent cause of discomfort, restlessness and crying amongst older infants. It is the skin manifestation of a sensitiveness to foreign protein and the protein which is reacted to, may enter the body through the medium of some biting insect, or through the ingestion of some food protein. Readjustment of diet and other treatment are discussed when dealing with skin diseases. The local application of dilute solutions of cresol or phenol is most effective in mitigating the irritation. These can be applied as baths containing ½ per cent to 1 per cent of the drugs or as lotions. A mixture composed of calamine lotion with 1 dram of glycerin, 30 minims of phenol, and augmented by 1 dram of zinc oxide or zinc carbonate, is very satisfactory.

Furunculosis is a common cause of distress to young babies whose nutrition is deficient. The back of the scalp is often the only region affected and before the boils have advanced to the purulent stage, they may easily escape attention, and the reason for the infant's crying and restlessness remain hidden.

The usual local treatment for furunculosis, together with attention to adjustments of the child's nutritional needs, will soon accomplish a cessation of the pain and crying. (See chapter on Skin Diseases.) Boils about the vulva and buttocks and on other parts of the body are less painful, although distressing, and they respond to the same kind of treatment.

Too much or too heavy bed clothing, or covers that are confining and restraining are fertile sources of discomfort and distress to young babies. Pressure on the immobile limbs, especially the feet, causes cramping and aching which is resented in

the only way possible, by whimpering and crying. *The insulating properties of large down pillows and soft mattresses* are causes of almost intolerable discomfort. Many unhappy, crying infants may be made tranquil by substituting firm mattresses, small, flat hair or floss pillows and light weight covers, loosely placed. *The child's clothing also may be uncomfortable.* Special attention should be directed to the fit and finish of arm bands, to the tightness of clothing that covers the chest, the wrinkling of ill-fitting diapers or those that are outgrown and make too much tension on the buttocks. The long flannel *abdominal band*, so popular with mothers and grandmothers, is especially to be condemned. It is wound several times about the belly and conscientiously pinned into place with the utmost snugness so that the ingestion of a feeding or the presence of any amount of gas in the intestine must increase the pressure unbearably. The humane horseman unloosens the girth on his horse before giving him a drink, but it is seldom that any one thinks of loosening the binder of a baby. There is no valid reason why a binder should be kept on a baby after the cord-stump has come away. If a band is used, it should never be made of an inflexible material but of some elastic fabric.

While overclothing, with consequent overheating, is a common source of discomfort, underclothing and chilling are also frequent causes of fussy discontent in babyhood. Thin babies, the prematurely born and weaklings, all are subject to excessive losses of heat and need especially to be protected from cooling. When crying can be related to such a cause, the remedy can be supplied by light, loose clothing and by the use of hot water bottles and heating pads and by a careful control of the room temperature.

CHAPTER VIII

CONVULSIONS AND SYNCOPES

One of the most alarming symptoms encountered in childhood is the convulsion. From the moment of birth until the end of the second year, such attacks are not unusual. The presence of a convulsion is always distressing, not only to the anxious parents but also to the attending physician, because the cause may run from some slight physiologic aberration, easily overcome, to a pathologic change of the gravest moment, in itself often essentially fatal.

To witness a severe convulsion is often disconcerting even to a practitioner of long experience and to the uninitiated, nothing can be more alarming. The staring, fixed, inexpressive eye, the change of color which may run from an almost deathly pallor in one case to an asphyxial cyanosis in another, the complete loss of consciousness and the rapid sequence of clonic convulsions, all combine to produce a distressing clinical picture.

There has been much discussion over the etiology of some convulsive seizures, although certain of them are referable to definite pathologic states. A great proportion seem to arise spontaneously. Furthermore, the conditions that cause convulsions in certain infants seem powerless to bring them about in others. From such facts as these, it is reasoned that there must be an underlying neuropathic tendency to convulsions, and family connections are searched to find epileptics, drunkards, narcomaniacs and other types of neuropathics in the lines of inheritance. Many convulsing babies have such a heredity. On the other hand, many such lines of descent show no infants subject to seizures, while some of the least neurotic family trees blossom with infants, victims of convulsive attacks. The recent discovery of salt content variation of the blood found in tetany is suggestive, and it may be the starting point of our knowledge of the etiology of these distressing seizures.

Convulsions were defined by Hughlings Jackson as "a sudden excessive discharge of many nervous arrangements representing movements, at once or nearly together, because the neurons subserving such movements have become highly unstable." Sta-

bility of neurons and nervous paths depend on maturity of the cells, on their proper nutrition and oxygenation, on complete myelination of the conducting apparatus and on freedom from toxic influences whether of autogenous or exogenous origin.

It follows then that the immature nervous system with its incomplete myelination is in a state of potential instability. Further the incompleteness of myelin insulation in the white matter of the brain allows an easy short-circuiting of nervous currents which can spread readily through the motor centers to produce clonic or tonic muscular spasms. Any reflex arc has two primary elements—a sensory and a motor. When abnormal sensations are maintained at an annoying pitch for some time and the nervous system is unstable from any cause, it is reasonable to suppose that the motor part of the arc may be unduly stimulated and by overflow bring about a generalized convulsion.

It is improbable that teething, or gastric irritation or the presence of undigested food stuffs in the colon with colic ever does bring on a convulsion through purely reflex irritation. However, should the assumption of reflex irritation be correct, the structure of the reflex arc may explain the phenomenon.

Circulating toxins, from diminished oxygenation as in *fever* and *asphyxiation* or from *imperfect nutrition; mal-adjustment of the internal secretions;* the *absorption of specific poisons* such as guanidine and histamine which may be elaborated in the gut, strychnine or tetanine that have been introduced from without; any or all of these are more likely to be the real etiologic factors in the reproduction of convulsions than is a reflex irritation.

Clonic convulsions asphyxial in origin may appear shortly after birth, because oxygen has been withheld from the neurons. Such a lack of oxygen in the blood may be due to pressure on the umbilical cord from one cause or another. As a result, the respiratory center may fail to begin its regulatory activities effectively, or perhaps there is some inability of the lung to expand. The convulsive phenomena may vary from a slight twitching of a few muscle groups to an extensive general clonic convulsion.

The reestablishment of respiration by artificial means, (see Methods), together with the use of hot baths or of alternating hot and cold douches to the lower chest and upper abdominal region will usually be followed by the prompt reestablishment of normal respiration and the cessation of the convulsion.

Unfortunately, but a very few of the convulsive seizures en-

countered postpartum are of this simple nature. The severe, intractable and often fatal seizures accompanied by convulsions may occur in the *offspring of mothers who have suffered from eclampsia* late in their pregnancy. The more severe cases of convulsions in such children, appearing immediately after birth are uncontrollable and fatal. However, in a certain proportion of them, the convulsion seems to be an anaphylactic phenomenon and occurs only after the first ingestion of breast milk. These attacks are of extraordinary interest and very unusual in appearance. The baby, apparently in perfect health and vigor, is put to the breast which it takes avidly. After a few minutes the child begins to breathe rapidly, becomes cyanotic, loses consciousness and enters into a convulsive state which may be limited to a few spasmodic twitchings or which may develop into a mild or severe convulsion. Most often these attacks are mistaken for the asphyxial cyanosis of debilitation with atelectasis. However, the differentiation can usually be clearly made through the fact that the atelectatic cyanotic attacks, with or without convulsive seizures, occur at any time and not always after the ingestion of food, as happens in the offspring of toxic mothers. Further confirmation of the diagnosis may be obtained on feeding the child on a properly devised artificial food, after which the convulsions cease. Once established on such feeding, no more attacks will appear. As in all other toxic states, it is wise to begin such a treatment by the use of a brisk purge, preferably at this age, 2/3 of a teaspoonful of castor oil with 1/3 teaspoonful of glycerin.

Mild asphyxial convulsions attack certain weakly babies who have an **atelectasis**. These attacks may occur very frequently during the day, as often as 30 or 40 times. The child will lose consciousness, turn cyanotic and exhibit some twitchings, though not with every seizure; or a well marked convulsion may appear.

As a rule these attacks are self-limited and the child comes out of them no matter what is done, and as these babies are asthenic, it is well not to attempt too much treatment. However, a mustard pack, mild artificial respiration and change of position are helpful in the most pronounced seizures.

At least one-half of the convulsive seizures that develop in the early postnatal period arise from definite **injuries to the brain** or from **malformations** of that organ. The injuries are for the most part due to *hemorrhages and lacerations* followed by fibroses

which may interfere with proper functioning of the neuron. Difficult labor may be a potent factor in the production of these hemorrhages and lacerations; but it is not nearly so frequent as is generally believed. Many cases occur in children born precipitately or during a normal labor and the obstetrician is incriminated many times without reason. The possibility that the child is hemophiliac or syphilitic must always be considered. There is a report of Warwick from the Department of Pediatrics of the University of Minnesota recounting postmortem examinations in infants dead of cerebral hemorrhages. Of these 44 per cent showed gross hemorrhages in other viscera; this indicates that hemorrhagic disease of the newborn may play a preponderant rôle. Rodda, working in the same institution, has found the coagulation time of infants who have hemorrhages at birth, to be markedly increased. These findings suggest that further investigation of the subject may be fruitful. Many of the cases of convulsions that occur soon after birth are found on postmortem examination to have been due to maldevelopment of the brain itself.

These groups of accidents—birth injuries, natal hemorrhages and maldevelopment of the brain—give rise to the cerebral conditions that in later infancy lead to the spastic motor disturbances: monoplegia, paraplegia, hemiplegia and diplegia. Even if only 25 per cent of these palsies result from prolonged and difficult labor, it would defend the position of these men who attempt a prophylaxis of spastic paralyses in the child through a Cesarean section of the mother. However, the morals of the situation should not be forgotten nor the fact that Cesarean section is not the harmless operation that it is often thought to be. Many conservative obstetricians are insistent in warning against the indiscriminate employment of the operation. Certain intracranial disturbances accompanied by convulsions may follow **emboli** arriving in the circulating system of the motor cortex, or **thrombi** that establish themselves in this region. In early infancy the common cause of emboli is infection about the umbilical cord, and later on in life, furunculosis, otitis media, epiphysitis and pyelocystitis are all causative factors that must be sought.

Aside from emboli, it must not be forgotten that **middle ear disease** and its complications, **mastoiditis and zygomatitis**, are often accompanied by an irritation of the nervous system, which may produce meningism, serous meningitis or lateral sinus thrombosis. The onset of any one of these affections may be ushered in by

a convulsion; therefore in the presence of a convulsion, the ear and mastoid region should be examined.

It has long been recognized that *syphilis* of the young infant is a fertile source of central nervous system changes, some of which give rise to convulsive seizures. True *syphilitic meningitis* is not very common. Jeans brings evidence to prove that a large proportion, if not all the cases of congenital syphilis of infancy, is accompanied by definite changes in the meninges and by alterations in the character of the spinal fluid.

The prophylactic treatment of the convulsion in such circumstances is treatment of the syphilis which consists essentially of inunctions of mercury and the intravenous injections of arsphenamine (salvarsan). (See Methods.)

Meningitis in infants is almost invariably ushered in by a convulsion, a symptom which appears very often many hours before other evidence of meningitis is clear. In the early weeks of life, the pneumococcus is often the infecting agent and then it is most often a terminal event in the course of a generalized septicemia. Frequently, arthritis, and epiphysitis are present as complications but under these circumstances it is rare to meet a true pneumonic consolidation. The staphylococcus, the streptococcus and various other bacteria are occasionally found in the meninges as etiologic factors of septic meningitis. Weichselbaum's diplococcus, (diplococcus intracellularis) appearing sporadically, or epidemically is the usual cause of meningitis in infancy. It is now generally realized that in this form of meningitis as well as any other, the inflammation of the meninges is but a part of a bacteriemia.

The *treatment* of the convulsive symptoms of the meningitides differs in no way from the treatment of convulsions of other origin and is given in detail at the end of this chapter.

In contrast to the early appearance of convulsions in the course of a diplococcic type of meningitis, is the late onset when the meningitis is of tuberculous origin. This form of meningitis is always a terminal event. For some weeks before any sign of meningitic involvement has appeared, malaise, malnutrition, headache, anorexia and low fever have been running on; so that the convulsion rarely appears as a revelation of this disease, as it so often does in the other forms of meningitis and in encephalitis. There is no adequate treatment.

It is often difficult to make the differentiation between an encephalitis and one of the meningitides at the onset of an at-

tack. Fuller discussion of this disorder and its differentiation is found in the chapter on Infectious Diseases. The convulsions that occur during the acute stage of an encephalitis are not the only ones for which this disease is responsible. Many of the convulsive seizures classed as **epilepsy** in later life can be attributed to brain damage through the scarring or cerebral atrophy that follows an *acute encephalitis.* Sometimes very marked and persistent convulsive seizures follow very mild attacks of nonsuppurative inflammations of the brain.

The relief of the seizures must depend entirely on specific remedies as it is unwise to handle the children more than necessary. However, a warm pack and the inhalation of a few drops of chloroform may aid in mitigating the distress of the child. (See p. 327.) The maltreatment that the "epileptic" receives at the hands of the laity is too well known to dwell upon.

Congenital syphilis sometimes causes a cortical arteritis that leads to patchy softening of and subsequent sclerosis of the brain tissue of young infants. This condition may give rise to convulsions. The earliest symptoms are usually slight unilateral twitchings of isolated muscle groups; later the whole limb may be affected; finally the convulsive seizure involves the entire body. In spite of adequate antisyphilitic treatment, spasticity and mental deficiency are the result for those who survive.

Besides the **exanthemata,** particularly *scarlet fever* and *measles,* other infections, notably *pneumonia,* may begin with a convulsion and reflex signs of central nervous system involvement. *Typhoid fever* and *malaria,* fortunately rare at this age, may also have the same kind of a dramatic onset.

Uremia with convulsions is almost unknown at this age. When it does occur, there has been antecedent *acute nephritis.* As a terminal event in the course of one of the fatal *liver diseases,* a convulsion may appear. Fortunately disease of the liver is an infrequent occurrence during early infancy.

The convulsive seizures of *pertussis* are truly of asphyxial origin in simple cases, and are due to mechanical obstruction to pulmonary oxygenation of the blood. There is reason to believe that in the severe forms of the disease, a poison is secreted which has a specific action on the neurons, increasing their instability. Cerebral hemorrhage is an accident that may occur in any pertussal convulsion and the resulting damage to the cortex may initiate further convulsions that may return throughout childhood.

Depending on the site of the bleeding, there may or may not be a residual spasticity of one or more limbs.

Convulsions in infancy may be due to *tetanus*. The common site for entry of the tetanus bacillus is the umbilical wound. Puncture wounds are occasionally responsible.

As a *prophylactic* following wounds which may possibly be infected by the tetanus bacillus, from 1000 to 3000 units of the antitoxin should be injected intravenously or intramuscularly. The dose is to be repeated on alternate days for 3 or 4 doses. Time is an important element. *Treatment* should be given early. If it has been delayed until symptoms are present, 10,000 to 18,000 units are to be injected intravenously and 5000 intraspinally, after the withdrawal of an equal or greater volume of spinal fluid. If the patient is completely anesthetized, success is more certain. If 2 or 3 c.c. of 5 per cent magnesium sulphate solution is injected into the spinal canal every day while the tetanic symptoms continue, the patient will be made more comfortable because of the muscular relaxation which follows such a use of magnesium sulphate. The injection of the antitoxin into tissues adjacent to the wound advised by some authorities is of doubtful efficacy.

A small proportion of children suffering from *rickets* develop convulsive seizures. Some of these attacks are of the ordinary clonic type, some are simple momentary tonic spasms. The greater number, however, are what have come to be known as **spasmophilic seizures.** These are characteristic. They rarely begin before the child is 9 or 10 months of age. The patient presents that clinical picture of malnutrition which is always part of a developing rickets. The attacks are of short duration. At their outset the child turns pale, falls over unconscious, sometimes uttering a cry. Often the seizure is silent. After a moment, twitchings ensue; these are often confined to the facial muscles, although any muscle group may be involved. Spasm of the glottis is almost always included in the manifestation and brings on a cyanosis. After a minute or two the child relaxes and resumes play without any more ado. Twenty or thirty such seizures may occur in a day of themselves, without having much effect on the child's health. Withdrawal of milk from the diet is promptly followed by the diminution in the intensity and number of the attacks and if this modification of the feeding is persisted in for some time the seizures will cease altogether. In addition, all the hygienic measures essential to the management of a case of rickets must be strictly

enforced. Amelioration seems to follow the use of phosphorus and cod-liver oil. Heliotherapy is paramount. (See Methods.)

Hydrocephalus that has followed an intracranial infection, prenatal or postnatal, may be a cause of convulsions. There is nothing characteristic about the convulsions but they are accompanied by the obvious physical signs of hydrocephalus.

In the presence of recurring convulsions, the possibility of a **brain tumor** must never be overlooked. The closest observation should be kept of such children for some time in order to determine the diagnosis. Not even the slightest sign that may aid in differentiation should be overlooked. It is true that brain tumors during infancy are rare, but it is equally true that the only chance of aiding a child who develops such a lesion is by an early diagnosis and by removal of the mass before too great a destruction of intracranial tissue has taken place.

Premature closure of sutures and fontanels with the production of pressure may be agencies of convulsion. This is particularly true in that type of case in which premature synostoses take place in the vertex while the cartilages of the base retain their function and so result in the production of the so-called *"tower skull."* The eyegrounds of such infants invariably give evidence of intracranial pressure and the peculiarity of the skull contour confirms the diagnosis. Ordinarily the premature closure of the sutures and fontanels leads to **microcrania** and means merely that the brain is congenitally incapable of attaining its normal size and function; that is to say, there is a **microcephaly** with microcrania. In the latter group of cases no means at our command can alleviate the underlying pathologic state or prevent the occurrence of the convulsions. In the "tower skull" variety of premature synosteal closure, on the other hand, operation is often brilliantly successful as the brain itself is uninjured. Unfortunately in this group of sufferers, only a few have the tower type of disturbance. The brilliant results that have been reported by surgeons working with remediable types have led to a widespread belief that any case of defective mind, especially if there be cranial imperfection, can be remedied by operation.

Under rare circumstances, **progressive degeneration of the nervous system** occurs in childhood and is a source, especially in the beginning, of convulsions which may puzzle the observer. However, the course of the disease during which there is rapid loss of mentality and increasing spasticity will finally elucidate the situation. The convulsions of such seizures are self-limited, of frequent

recurrence and often of an intensity that increases for some time, after which they disappear.

Treatment of the Convulsion.—The convulsion is a self-limited seizure and without treatment usually will terminate spontaneously quite as promptly as when the common methods of treatment such as hot baths and enemata are used. The value of hot baths and enemata is largely in their power to alleviate the anxiety of the family and attendants and to occupy their minds while the seizure is following its normal course. However, in those cases in which fever is a factor in producing cerebral irritation, packs and baths given in order to reduce temperature are promptly effective. When cyanosis is persistent, the mustard pack through its stimulation of the sensorium may be of great assistance.

When the instability of the nervous system is very great and the convulsions repeat themselves in rapid succession, the use of drugs is indicated. The best drug under these circumstances is chloroform given by inhalation. It has been objected that chloroform does not reach the lungs during a convulsion because the patient is not breathing. However, anyone who has used the method knows that a very small amount of chloroform given when the child commences to twitch preliminary to the beginning of a seizure, will in many instances prevent the full development of a convulsive attack.

Years ago, a case of strychnine poisoning came to the observation of one of the writers. The patient was kept partially narcotized with chloroform for 24 hours and made a perfect recovery. Since that time, another child in his practice developed severe symptoms of strychnine poisoning. This child was also kept under the influence of chloroform by relays of anesthetists—in this instance, for thirty-six hours. It is only in cases where the convulsions recur at short intervals that the use of chloroform is indicated.

It is essential to remember in such cases that strychnine is excreted into the bladder and is reabsorbed so that frequent catheterizations are necessary.

Chloral is an effective drug but it must be given in sufficient dosage. Given by rectum in warm water, in doses of 3 grains for a 15-pound child, with or without 5 grains of sodium bromide, it is absorbed rather slowly and its effect is delayed for some time. It can only be efficacious in mitigating the irritability of the unstable neurons.

Altogether the most effective drug we have for control of the course of recurring convulsions is morphin, used hypodermically. In such an emergency, $\frac{1}{20}$ of a grain may be given for a 15-pound baby. Hyoscine hydrobromate has been recommended but it is mentioned only to be condemned. It is a very dangerous and uncertain drug.

The use of purgation is a time-honored and apparently harmless procedure and there may be advantage in giving a dose of 2 or 3 drams of castor oil or a grain of calomel as soon as the child is able to swallow. A high enema will often bring away masses of undigested food to which are often attributed an etiologic function in the production of the convulsion; but if one washes the bowel of a well child, similar masses may be discovered. The great advantages of such a procedure lie in the reduction of fever if any be present; and when there is distention of the bowel with pressure on the diaphragm an enema is of advantage.

CHAPTER IX

FEVER

No single physiologic fact more decisively marks the difference between the infant and the grown, than the instability of the baby's heat regulating apparatus. Both hyperthermia and hypothermia are frequently encountered during infancy. While not a proved fact, it seems probable that the delicate adjustment of the infant's water balance is the basis of the ready fluctuations that the infantile organism undergoes. It is certain that the characteristic rises in temperature, encountered so frequently during the first few days of life, follow water impoverishment incident to the **starvation** period that precedes the establishment of the mother's colostrum flow.

The same type of fever may be met at any time during the first two years as a result of **dehydration** from diarrhea or from any other cause. It apparently depends on the loss of water available for the rapid evaporation from the lungs and skin. It has been suggested that a sufficient water intake might prevent hyperpyrexia even in those intoxications and infections which are accompanied by great rises in the body's thermal output. That the balance of water metabolism is an influential agent in the production of fever is borne out by those experimentalists who have shown that fever can be produced by the injection of hypertonic salt and sugar solutions, substances that tend to immobilize water in the body and to prevent its ready evaporation.

Beside starvation and dehydration mentioned in the preceding paragraph, **infections** give rise to conditions which are made evident by sharp rises in temperature. The infections which are to be incriminated when a newborn baby is persistently feverish are to be found for the most part, about the umbilicus.

Early diagnosis and treatment of *umbilical infections* is essential, as ascending involvement of the umbilical vein readily reaches the liver with the production of an *infective perihepatitis* or at times even a *cholangitis* or a *generalized peritonitis* may occur without any pain, tenderness or distention to call attention to the involvement of the peritoneum. From either a peritonitis or cholangitis, general *septicemia* may rapidly develop and prove fatal.

The *treatment* for these conditions is essentially preventive. Scrupulous care in handling of the umbilical stump should be observed. Once there is infection, prompt cutting away of the necrotic tissue and the free application of iodine in glycerin is indicated. When a peritonitis is discovered, immediate and thorough surgical intervention is the only aid to recovery.

With the newborn, occasionally an infection of the *middle ear* or an inflammation of an *epiphysis* will be the cause of fever. However, in such a case, pain usually will be the predominant symptom. The treatment should be directed toward the cause.

In the case of infants beyond their first month, the two common causes of obscure fever are *middle ear infection,* and in girls *infection of the genitourinary tract.* The middle ear may be inflamed and produce high fever without any accompanying pain to call attention to it. A thorough examination of these sources should never be neglected even when there is other obvious cause for fever.

Although the patient be a male, search of the urine should not be delayed although cystitis is very rare and pyelitis uncommon in the case of boy babies. Once the infection is conquered, the fever will subside. It may often be necessary to use hydrotherapy as an antipyretic measure, for in no disease of infancy does the temperature range higher or persist more tenaciously than in cystitis. For details of treatment see the chapter on the Genitourinary Diseases.

Acute *tonsillitis* with *pharyngitis* as a cause of pyrexia is well known. Perhaps it is the pathologic state most frequently followed by fever in infancy. Many little patients who are purged and drugged because it is supposed that the intestine is at fault, are in truth suffering with a fever that follows bacterial invasion of the fauces. Most of these throat infections are due to cocci. Fortunately, B. diphtheriæ is a rare invader during the first two years. Either alone or as a complication of infected throats, *laryngitis* may originate a fever. Most often, however, this involvement is but slight and frequently there is no rise in temperature above the normal. The fever in such cases rarely needs special consideration.

When the lower respiratory tract is involved, the symptoms of that involvement will be obvious and only occasionally will the fever as such, be troublesome. When it is so in instances of *bronchitis or bronchopneumonia,* mustard packs are effective both for

reducing temperature and in soothing the cough. In *lobar pneumonia,* there will be but few cases in which treatment of pyrexia will be urgently needed. Simple spongings or tepid packs will most often suffice. When pain is a complication, mustard packs may be used for their analgesic as well as their antipyretic effect.

Except in infections and infectious diarrheas, *the intestine* is rarely the source of high fever, although it is undoubted that a single drastic purge is often followed by a decisive drop in the temperature. Hydrotherapy and subcutaneous, intravenous or intraperitoneal injection of water are indicated in those cases of pyrexias of intestinal infections such as occur in the *dysenteries, typhoid* and *paratyphoid.*

The infectious diseases attack infants rarely. *Pertussis,* in which fever is seldom high enough to give concern, occurs rarely during the early months of life. Late in the first year and during the second year of life, *measles* may be a puzzling source of fever. Sometimes as many as 6 or 7 days of fever may precede the appearance of the coryza, buccal rash and injection of the inner canthi. After this, fever is high, ranging in many cases to 105° or above.

Other *blood stream invasions* by known or unknown bacteria accompanied by pyrexia occur during the first 2 years. Most often immunity is promptly gained and the fever disappears after a few days. When such a fever persists, blood culture is in order and if the technic is correct, in many instances, the infecting organism will be revealed. Under these circumstances, hydrotherapy is the *treatment* of choice for the reduction of the temperature. For fevers of low degree, that is from 102° to 103°, tepid sponges and immersion baths are to be preferred. When the temperature reaches 103° to 106°, then packs, tepid or hot, are more effective. When respiratory disorders are to be dealt with, mustard packs are remarkably useful. Cold baths and cold packs are out of place. The old fashioned Preitznitz bandage, changed every 4 hours, may be of value when the temperature fails to respond to tepid or mustard packs. Details of methods for preparing and using baths, packs and spongings are given in the chapter on Methods.

Whether drugs shall be used for their antipyretic effect is a question difficult to answer. In view of the effectiveness of hydrotherapy and its flexibility and simplicity as a therapeutic measure, it seems to us that antipyretic drugs, as such, well might be abandoned. If it is desired to use such remedies, however, the

fact that most of them are heart depressants must not be overlooked, and prescriptions containing them should include camphor, or caffeine-sodium-benzoate. Antipyrin has the merit of relative tastelessness and a certain amount of analgesic power. Combined with ½ grain of caffeine-sodium-benzoate, 1 grain of antipyrin may be given to a 15-pound baby every 4 hours for 6 or 8 doses without harm. If the patient has passed the first birthday, pyramidon, 1 to 1½ grains in powder with sugar every 4 hours is a more effective analgesic, especially when headache is severe. It is an equally potent antipyretic. There are other antipyretic drugs available, none possess any properties that make their use of advantage in the treatment of the symptom fever as it occurs during infancy.

CHAPTER X

COUGH

Cough may arise from irritation anywhere in the upper respiratory tract or in the external ear. **Foreign bodies** are often offenders. A persistent cough with a one-sided, purulent, nasal discharge may indicate a button or a bead in the nose. The *treatment* is the removal of the source of irritation.

Nasopharyngeal myxoma or **adenoids** are frequent sources of dry, unproductive cough. So also is the viscid secretion of a **nasopharyngitis**, especially in young babies. The *treatment* of the former is removal; of the latter is painting with a 5 per cent silver nitrate solution followed by demulcent drinks such as sweetened flaxseed tea flavored with orange juice.

A persistent, irritating cough is one of the early prodromal signs of **measles**; continuing after the eruption is well developed, it indicates pulmonary complications. (See page 439 for treatment.)

In the presence of a persistent, brassy cough of acute onset and increasing intensity, especially if urgent dyspnea supervenes, with over use of the accessory muscles of respiration, and infall of the supraclavicular and infraclavicular spaces and lower ribs is observable, **laryngeal diphtheria** may be suspected. A sufficient dose of antitoxin should be given even before microscopic diagnosis is possible. (See page 430.) In the face of such a cough with increasing cyanosis and dyspnea, intubation must not be delayed even though Klebs-Loeffler bacillus cannot be demonstrated. (See page 560.)

Not all brassy, laryngeal coughs indicate laryngeal diphtheria; the cough of **croup** from simple catarrhal laryngitis is common. It is characteristic of this condition that the cough is absent earlier in the day, comes on toward evening with increasing intensity and is most severe during the night. Cyanosis and dyspnea are usually absent except during the paroxysm of coughing. Even without treatment, improvement comes on in the morning. In many cases the cough is self-limited and lasts only 2 or 3 days. Usually croup is a complication or a sequel of rhinitis or acute pharyngitis and tonsillitis. However, removal

of the tonsils does not always prevent future attacks. The *treatment* is dealt with on page 173.

Tracheal cough is usually accompanied by dyspnea; it is less strident and more painful than laryngeal. The *treatment* is relatively that of laryngeal croup with the addition of light mustard applications to the chest. Heavy packs are to be avoided. The paroxysmal attacks of coughing which occur in whooping cough, are essentially tracheolaryngeal in nature.

In very young babies, severe, persistent, strangling cough, often without whoop or vomiting, may indicate **pertussis.** (For *treatment* see page 461.)

A cough of similar character but less severe, accompanied by inspiratory dyspnea should cause suspicion of an enlarged **thymus** gland. The diagnosis may be confirmed by a well-taken radiograph. The symptoms of enlargement of the gland will usually decrease after 4 or 5 exposures to the x-ray in amounts of ½ an erythema dose given at 10 day intervals. (See page 399.) Radium emanations have also been used with success.

The cough of **bronchitis** as it affects infants, is persistent and harassing when the medium and small tubes are infected. When the larger tubes are involved, there is usually a complicating tracheitis and the cough is painful. Cough is a protective reaction and it is to be encouraged rather than checked unless it becomes so disturbing that rest is interfered with. (For *treatment* see page 178.)

The cough of **bronchopneumonia** is unproductive, painful and therefore, shallow. In infants the inflammation is of the finer bronchial tubes as well as in the air cells which tend to consolidate in scattered patches. At this age spasm of the medium-sized bronchial tubes is a feature of the disease. These facts give the indication for treatment, which is to relieve the spasm and support the circulation. (See page 180.)

Beside bronchopneumonia and capillary bronchitis, many infants suffer from recurrent attacks of **spasmodic** or **asthmatic bronchitis** brought on by sensitization with proteins ingested or inhaled. Pollens, foods, animal emanations and bacteria may furnish the offending protein. The cough is incessant, shallow and distressing. It is almost as characteristic as the wheezing respiration or the dyspnea pathognomonic of asthma.

Irritative and frequently painful cough, may be the only sign except increased respiration and heightened temperature, to in-

dicate the onset of a **lobar pneumonia** in infancy and even in later childhood. Often the crisis has taken place before the auscultatory and major percussion signs appear. In such an instance, diagnosis must be made by exclusion, and the cough is a valuable aid. Especially if it is combined with a decreased movement of one side of the chest during a coughing attack.

Similar findings with the same type of restrained, painful, shallow, unproductive cough occur when a **dry pleuritis** affects the pleura. On auscultation the listener may discover many dry clicks. Such a cough will disappear spontaneously. The condition undoubtedly explains many of the mysterious coughs of infancy and childhood. Effusion can follow such a dry pleurisy even in babies. Its first indication is the development of unilateral immobility. Prompt aspiration will usually bring complete relief. (See page 203.)

CHAPTER XI

PREMATURITY

Prematurity causes many of the deaths which occur within the first 10 days after birth, and it is responsible for the major part of those ill-nourished infants who later suffer from constitutional inferiority expressed in poor heat regulation, inefficient respiration, vascular instability, anemia, rickets, asthenia, impaired digestion and susceptibility to infection.

The vulnerability of the child will depend on the cause of the early birth. In general the shorter the time between conception and birth, the less vigorous the premature. Maternal diseases, especially syphilis, sepsis, and tuberculosis, render the child's chance of survival less, and in proportion to the severity of the mother's infection. Otherwise, the prognosis will depend on the time elapsed from the date of conception, recorded in days, on the circumstances of the delivery, on the opportunities available to the physician for maintaining the infant at a temperature which will prevent undue heat loss, and on the chance of obtaining breast milk.

In his invaluable book, "Premature and Congenitally Diseased Infants," Julius H. Hess records that the lightest premature infant known to have survived, weighed 500 grams (1 pound, 1 ounce). General experience is, however, that of infants born with a weight less than 2000 grams (4 pounds, 4 ounces), not more than half can be expected to live; while of those weighing up to 2500 grams (5 pounds, 4 ounces) three-fourths ought to live. In the heaviest group (over 5 pounds at birth), the mortality rate differs but little from that of average weight, full-time babies.

In reality weight is less prognostic than age. Up to now, accurate computation of the premature's age has been impossible; but thanks to the radiographic method devised by Hess, in future we will be able to infer the approximate age from reading a well taken x-ray plate.

As a prognostic criterion, the hypothermia of the child and its inability to maintain a rectal temperature above 92° are of equal significance with the low weight. Well favored and capably

managed prematures have survived with a temperature at sometime as low as 90° F. It matters little whether the hydrothermia is a result of intracranial hemorrhage, or itself is directly a cause of debility, the need to maintain a temperature of about 80° in the atmosphere surrounding the child is imperative. Slow acquisition of normal body heat, once the child is in an incubator or warm bed, indicates a poor chance for survival.

As important to consider in forming a prognosis, is the character of the premature's respiration. The more perfectly air enters the lungs the more rapidly, other factors being favorable, can we expect the child to become normal. Attacks of cyanosis may indicate no more than functional immaturity of the nervous system. The cyanotic attacks often seen in prematures of low weight are of this nature. Very often, however, these are complicated by the persistence of fetal atelectasis, for the lack of irritability of the immature nervous centers limits the number and weakens the force of the nervous impulses needed for the proper action of the poorly developed respiratory muscles.

While cyanosis may be simply a manifestation of immaturity, there are other causes which may interfere with the respiration and bring about cyanotic attacks and even strangulation. Aspiration of maternal discharges at the time of birth or of ingested fluids or of vomitus afterward is especially likely to be causative of strangulation with cyanosis.

Dehydration from insufficient fluid or food—too large or too frequent meals, with acute, flatulent distention of the stomach or intestines, rarely a diaphragmatic hernia—may explain cyanotic attacks and respiratory failure in the premature. True congenital cardiac deformity may cause cyanosis but the inefficiency of cardiac or respiratory muscles or the persistence of atelectasis singly or combined, may hinder the closure of the ductus arteriosus and the foramen ovale and interfere with pulmonary circulation.

Inadequate respiratory powers render premature infants easy victims to pulmonary disorders—pneumonia, bronchitis, atelectasis and especially edema. It is therefore imperative to shield the infant from contact with persons with respiratory infections, however slight.

Among the other deficiencies of the premature is the weakness of its digestive system. Anorexia, vomiting, diarrhea and abdominal distention are frequent complications, often to such a degree as to constitute the clinical condition known as food

damage. This is more apt to come on when the supply of human milk fails. Often such a state is exaggerated because the congenital debility of the infant leaves him continually drowsy or sleeping, or if wakeful, uninterested in sucking.

The effective *treatment* of the premature depends on preventing undue loss of body heat, on the avoidance of cyanosis and asphyxia, on gentle handling, on the provision of adequate fluid supply and on the provision of a food proper in quality and quantity.

The delivery room should be *heated* to about 80 degrees. If artificial respiration of the newborn infant is necessary, the manual manipulation should be done with the child wrapped in a warm blanket. An effective method of making artificial respiration is for the assistant to support the infant by holding it by the legs and back of the head and in an inverted position; the operator enfolds the chest with both hands, thumbs behind and the fingers spread out in front, and makes gentle rhythmic compression at the rate of about 20 per minute. Once respiration is established, it is better to depend on warmth and the administration of warm oxygen (see page 556) to control the cyanosis and asphyxia. The infant should not be uncovered again until it is slipped into the premature robe, which may be one specially prepared or an emergency garment quickly gotten together. This latter can be improvised with a sheet of cotton laid between two layers of soft cotton cloth. A smaller square can be attached to the top to act as a hood. In dividing the cord, exposure must be avoided. Without a bath and so clad, the child is to be rapidly placed in a warm chamber, where the temperature can be maintained at 78 to 83° F. This chamber may be a heated bed, either of the specially devised Hess type, a portable incubator or an improvised container. Whenever it is possible to reach an institution which has special equipment for caring for the premature, the infant should be placed in its protection at as early a moment as possible. Without any doubt the mortality of prematures could be greatly lowered if this advice could be followed.

Failing to obtain special incubator apparatus, a substitute can be contrived by putting two bricks into the bottom of a box of suitable size; between these may be placed an ordinary deep roasting pan, into which a hot water bottle or, preferably, an electric heating pad is placed. On the bricks and over the pan, but not too close, is laid a well-padded small basket to contain the

baby. The mattress should be large enough that the infant may lie high in the basket. A special hood may be devised to retain heat and insure ventilation, and a blanket placed over the remainder of the box.

During the earlier days of the child's life, this warm bed should be kept in a room in which the temperature is not permitted to drop below 70°, and in which the humidity is well maintained by the constant evaporation of water, placed near the crib or by hanging up wet sheets or towels. Gradually the temperature of the bed and of the surrounding room should be lowered until at the end of a period, varying with the vitality of the child from 6 to 16 weeks, the infant is subjected to the usual bed and room temperature. The change should be made gradually, for a sudden withdrawal subjects the baby to hypothermia and the chance of cardiac, respiratory and digestive failure.

A free supply of fresh air is essential to the well being of such infants. Although the oxygen consumption is small, it is imperative that the room in which the child lies should be kept for it and its attendant alone. *Ventilation* should be insured by open windows in an adjoining room. If this room can also be used solely for the infant, there is added advantage, particularly if it be a bath room.

At all times, even during the later stages of its progress, such an infant loses heat rapidly. The clothing should be warm and loose. The usual flannel band is objectionable; it constricts the abdomen and interferes with breathing. Soft cotton should be worn next to the skin and the outer garments should be wool.

As premature babies are prone to develop *asphyxia,* prompt reduction of the cord, if it should prolapse during birth, is of the utmost importance. Rapid completion of delivery may become necessary if it is apparent that asphyxia is impending. The removal from the nose, mouth and pharynx of any maternal discharges that may enter is necessary. Such a clearance is readily made by aid of a small catheter attached to an aspirating bulb. When attacks of cyanosis are frequent or severe, especially if complicated by hypothermia, the use of an occasional mustard pack is valuable. The small blanket for the pack is to be wrung out of a solution made by combining two teaspoonfuls of ground mustard mixed with cold water, and one gallon of hot (not boiling) water. The child can be slipped into the pack without undue exposure.

The maintenance of body heat and an adequate *fluid supply* is of utmost importance in the prevention of cyanosis. Every twenty-four hours the premature child needs an amount of fluid equal to one-fifth of its body weight. If the intake falls to a quantity less than one-seventh of its body weight, the child will die. Very few premature babies in the earlier weeks of life will take such amount of breast milk. Therefore the difference between the milk intake and what they require must be given in the form of water or sugar solution. This excess may be divided into 3 or 4 portions and given from a syringe by rectal instillation if necessary.

Without *mother's milk* feeding, the death rate is appalling. Every effort must be made to provide human milk from other sources before the mother's milk, the appearance of which is often delayed, is established. The premature child's attempts to swallow often bring about respiratory failure; therefore, no feeding should be given for the first twelve hours in order to allow this function to become better established. During the first days of life, small amounts of human milk (at least one-tenth of the body weight, preferably one-eighth) should be fed to the inert and weakly infant; this should be given through a catheter in 3 to 5 daily feedings. Then the feedings should be limited to an amount equal to about 1 ounce per pound per day. This amount should be increased at the rate of ½ to 1 dram per day until the optimum feeding of 2½ ounces per pound per day has been reached. If the child is doing well, the increase may be made more rapidly at the end of ten days or two weeks. However, not more than 1½ drams per day should be added even for strong prematures.

The *feeding interval* will depend on the size of the meal the child can accept. Weak prematures who cannot take more than one-third to one-half teaspoonful at a time will have to be fed at shorter intervals; they should receive from 8 to 10 meals a day when the dropper is used, or 4 to 6 when a catheter is employed.

The mother of a moderately heavy premature baby will probably be able to nurse her offspring after the first few days. She should be encouraged to attempt the development of a milk supply. It is especially important that the breasts be entirely emptied at least 4 times in twenty-four hours; this may be done by manual expression or preferably, by an infant, other than her own, who can nurse vigorously. Breast pumps are objectionable.

Strong prematures should be allowed to suckle. For those less favored, the milk should be expressed and fed with a rubber-tipped medicine dropper (see page 574). It is possible to express the milk directly into the child's mouth; the disadvantage lies in the ease of overfeeding.

The very weak must be fed by catheter. On a No. 12 French, No. 8 American, soft rubber catheter the distance from the lower end of the catheter laid on the ensiform cartilage to the bridge of the nose, should be indicated on the rubber with a file mark. A small funnel is mounted on the catheter and with the child recumbent, the tube is passed until the file mark is at the lips. The food should be poured in slowly, after which the tube should be pinched and sharply withdrawn.

Failing mother's milk, artificial feeding must be employed. The writers prefer whey-cream mixtures. The same principles regarding fluid and caloric intake prevail as in feeding mother's milk. It is always highly desirable that when possible, at least a small amount of mother's milk be given.

The whey-cream mixture is prepared as follows: take 3 ounces of the top 5 ounces of a quart of milk on which the cream is well risen. To this add 2 ounces of skimmed milk, 10 ounces of whey (see page 592), 5 ounces of water, and ½ level tablespoonful milk sugar. As the child grows the skimmed milk may be increased at the expense of the water at the rate of ½ ounce per week.

Some authors advise lactic acid milk modified with dextrinized flour and cane sugar. This can be prepared for feeding in the early weeks as follows: take 16 ounces of fat-free lactic acid milk (see page 590); 2 level teaspoonfuls of dextrinized flour (flour ball, see page 598), or Imperial Granum; and 4 level teaspoonfuls of cane sugar. For the later weeks the flour ball may be increased to 3 teaspoonfuls and the sugar to 5 teaspoonfuls. The paste is made with the flour and a small amount of cold milk. The remainder of the milk is added and the mixture brought to a boil. It is then taken from the fire, the sugar is added and again returned to boil. The volume is brought up to a pint with cold, boiled water and the mixture set on ice. During the first month of life, fat-free milk is used in making the lactic acid milk; later the lactic acid milk should be prepared from a mixture containing one-third whole milk and two-thirds skimmed milk. After the second month equal parts whole milk and skimmed milk should be used in its preparation.

PART II

CHAPTER XII

DISEASES OF THE RESPIRATORY TRACT

Certain infants are born with **congenital nasal atresia** due to the persistence of a diaphragm in the posterior part of the nasal fossa. The obstruction may be unilateral or bilateral. Early in life the diaphragm is membranous; later on, bony. The obstruction leads to mouth breathing, and to this is added a constant discharge of mucus from the anterior nares. Excoriation of the lip appears because of the constant flow of nasal secretion. Such a clinical picture may simulate syphilis. During the first months of life, the membrane can be removed without difficulty by a simple incision; later, it will be necessary to use bone-cutting instruments, and sometimes it may be wise to remove the posterior part of the vomer as well.

Coryza is a common manifestation in infant patients. The condition is due to a combined swelling of the mucous membrane of the nose and the appearance of a hypersecretion caused by bacterial, mechanical or chemical agents. There are also certain instances in which vasomotor instability leads to coryza. During infancy by far the commonest source of the irritation is bacterial, and any of the cocci may be etiological. The micrococcus catarrhalis is especially active in the coryzas of early life, and there are certain bacilli which may be often incriminated. Coryza is also a prodromal symptom of measles and lower respiratory tract infections as well as a part of the clinical picture of syphilis.

The onset of simple coryza in infancy is marked by lassitude, nasal obstruction and a slight rise in temperature, rarely to a point above 102°. The breast or nursing-bottle is badly taken, sleep is disturbed and in young infants an alarming attack of dyspnea may occur from no cause other than nasal obstruction. The delicate vascular structures in the nose may rupture and the discharges become sanguinous. This is especially true when the Spirocheta pallida is the microbian cause of the disorder.

Almost invariably, the nasopharynx is involved with the anterior nares, and under such circumstances, it is common for the inner ends of the Eustachian tubes to become swollen and occluded with the result that negative pressure on the ear drums causes their retraction and an earache. In many cases, not only the inner end of the tube but also the whole extent of its mucous membrane becomes involved in the inflammatory process. This inflammation is carried on to the middle ear and an otitis media results. Often the pharynx, larynx and bronchial tubes are invaded by extension during the progress of a microbian coryza.

When an irritant is the cause of the coryza, the onset of the disorder is remarkable for its suddenness. Exposure to formalin gas or to some other irritant vapor, the insufflation of talcum powder, or sometimes the local application of adrenalin may set up a rhinitis. If an infant is susceptible to pollens, which are quite the commonest irritative causes of coryza, the presence of plants or cut flowers in the nursery may be etiologic; potted primroses especially may account for cases of puzzling paroxysmal coryza. The pollens rarely cause coryza only. The nasal manifestations are usually accompanied by a conjunctivitis. The secretion produced by these irritations is more watery and less cellular than when bacteria originate the coryza.

The *treatment* of coryza, if its cause is mechanical, is prophylactic; if microbian, it is essential that the patient be placed in a warm, well ventilated room in which the air should be kept moist. The mustard bath (see Methods) given at the onset of the symptoms may prove useful. Properly done, irrigation of the nose, when carried out early, occasionally will abort a coryzal attack. The irrigation should be followed by the instillation of an oily solution composed of adrenalin (1-3500) minims 30, grey oil minims 30, liquid petrolatum drams 2, a few drops to be instilled into the nostrils with a medicine dropper. Atropin 1/2000 grain in watery solution, may be given every 2 hours for 3 or 4 doses; this drug may aid in aborting an attack.

Membranous rhinitis is not often seen during infancy. It is almost invariably an index of diphtheritic involvement of the nares. The proper *treatment* is to isolate the patient and to inject antitoxin.

Chronic rhinitis is the frequent sequel of a number of successive attacks of acute rhinitis. It most often occurs in those chil-

dren who have hypertrophied adenoid tissue obstructing the nasal passage.

The *treatment* is the removal of the adenoid and the subsequent systematic cleansing of the nose by spraying or syringing with a mild alkaline solution. (See Methods.) This should be followed by the daily instillation of ½ of 1 per cent yellow oxide of mercury ointment. The use of adrenalin should be avoided when dealing with chronic rhinitis.

Acute tonsillitis is a morbid process that happens with great frequency during the first 2 years of life. Because it often occurs at this age without apparent pain and with little evidence of difficulty in swallowing, it may be overlooked, and a great many children who suffer from tonsillar infections are treated for gastroenteric disorders or for bronchitis. Bacterial invasions about the fauces very often produce fetid breath and a coating of the tongue, signs popularly interpreted as evidences of indigestion, but which in truth, are almost always the result of tonsillitis or of pharyngitis. The disease is contagious and may be caused by the implantation of bacteria transmitted from an individual suffering with an infection in any part of the respiratory tract. Coryza or bronchitis in a parent may set up an infection in the child which will develop into a tonsillitis; on the other hand, the contagion of the tonsillitis may be passed on and cause a tonsillitis, a rhinitis or a pneumonia in some one else. Some epidemics of tonsillitis have been traced to the ingestion of milk contaminated by streptococci, and often such a streptococcal tonsillitis is the precursor of a *nephritis,* of a *septicemia,* or of a localized purulent process—an *arthritis,* an *osteomyelitis* or a *meningitis.*

Acute tonsillitis of moderate or severe type forms a part of the clinical picture of scarlet fever. In measles also, the tonsils are swollen as part of the upper respiratory tract tumefaction and reddening. The appearances of tonsils involved in a faucial diphtheria are characteristic. The condition is treated fully in the chapter on infectious diseases. The exudate in a simple follicular tonsillitis is patchy and appears at the opening of the crypts; it tends not to coalesce, and it may be removed without causing any bleeding of the underlying mucous membrane. However, the two conditions may present appearances extremely difficult to differentiate, and a final judgment must await results of cultures taken from the exudate and grown on

blood serum. In case of doubt, *the prompt use of antitoxin* can do no harm.

The fever curve of a tonsillitis usually ranges higher than that of a diphtheria, and the toxic symptoms of the affection are less an expression of stuporous intoxication than of stimulation. The infant will be restless, tender to the touch, and it will cry from spontaneous muscular pain. A great deal of inflammation of the tonsils may occur with redness and swelling without much interference with swallowing. On the other hand, in many cases there is a great deal of tenderness on deglutition, and the child may refuse to take its food. This is especially likely to happen when the cervical glands are also swollen and tender.

Local *treatment* is indicated in all cases. If the patient is seen early, the application of 10 per cent silver nitrate painted onto the swollen tonsils is of distinct value. Repeated applications are unnecessary; one or two paintings suffice. Instillation through the nostrils of a mixture composed of adrenalin (1-3500) minims 30, grey oil minims 30, petrolatum drams 2, will prove of advantage. The salicylate of soda, grains 2, bromide of soda, grains 2, in sweetened water dram 1, given every 3 hours for 12 to 14 doses is helpful. A mixture of bichloride of mercury grain 1/1000, tincture chloride of iron minims 5 to 10, glycerin minims 15, every 2 hours for 5 to 6 doses has been recommended.

The sickroom should be kept well ventilated at about 65° F. The use of the steam kettle is sometimes an aid to treatment. The application of a wet pack to the throat of older infants seems to be a comforting procedure, and when the swelling and pain is great, irrigation of the fauces with hot alkaline solution is followed by much relief.

When painting, spraying or irrigating the throat, it is essential that the child should be restrained in a properly applied sheet or blanket. (See Methods.) Without such restraint, the struggling of the infant makes it impossible to carry out the procedures properly. The child's efforts exhaust its strength as well as the patience of the physician.

Vincent's angina is occasionally met with during infancy. The disease is a result of infection by a fusiform bacillus growing in symbiosis with a typical spirochete. As a rule, the disease attacks one tonsil only. It produces a white membrane that may readily be mistaken for the membrane of diphtheria. Beneath this the surface of the mucous membrane is ulcerated rather

deeply. The ulcers are indolent with little tendency to heal; they may occur not only on the tonsils but anywhere in the mouth. Single ulcers have been mistaken for primary lesions of syphilis. A smear from the surface of the ulcer showing the characteristic spirillæ and bacilli will render the diagnosis positive. The constitutional symptoms are slight and the fever is of low degree.

Iodin is exceedingly effective in the *treatment* of this disease. Lugol's solution or 10 per cent iodin in glycerin and water applied once or twice a day brings about a rapid healing. Arsphenamine has also been used with success as a local application to the ulcerated surface.

An **adenoid** is a normal lymphoid structure which grows on the roof and the back wall of the nasopharynx. Certain children are born with highly arched palates and small capacity of the nasopharynx. Even with a normal amount of lymphoid tissue, such children may have so marked a nasal obstruction that nursing is difficult; therefore, even in very early life, it may be necessary to curette the nasopharynx to remove the obstructing lymphoid mass. The contour and capacity of the nasopharynx of the Mongolian idiot is such that a very small amount of adenoid tissue will produce a nasal obstruction.

When the size and shape of the nasopharynx is normal, a series of attacks of nasopharyngitis with infection of the lymphoid tissue may give rise to a congestion that leads to marked hypertrophy. This hypertrophic tissue then constitutes what is known as an adenoid vegetation.

The most striking symptom is the nasal obstruction with consequent mouth breathing. The facies of patients with adenoid obstruction is characteristic. The nostrils are narrow, the nose is pinched, usually the palate is high, and the alveolar arch is flattened. The obstruction to breathing is not constant in all cases. It may vary from time to time with the amount of congestion in the adenoid tissue. Recurrent rhinitis is a common complication, and the nasal inflammation tends to persist. The growth may occlude the inner aperture of the Eustachian tube, and as a result partial deafness and attacks of earache are often encountered in a patient with adenoids. The commonest cause of otitis media is this obstructive effect of the adenoid on the Eustachian tube.

Not only the ear but the larynx, trachea and bronchi seem to be rendered more vulnerable in patients who suffer with adenoids.

Much exaggerated stress has been laid on adenoids as a cause of mental deficiency. The removal of these growths will never restore mentality, nor will it ever clear up an asthma, nor cure a nocturnal enuresis. The only *treatment* for adenoids is their removal, followed by heliotherapy and a supervised regime.

Almost always **simple hypertrophied tonsils** are the accompaniment of adenoids. Their enlargement is ordinarily of no consequence and presents no symptomatology. The *removal of tonsils which are neither obstructive nor infected merely because they are large, has no place in modern therapeutics.* When the enlargement is the result of frequently recurring infection, the case is different, and tonsillectomy is often followed by gratifying results even when the organs are removed as early as the second year. There seems to be no authentic evidence that the tonsil is an organ of internal secretion, and therefore there is no contraindication to the removal at any age of tonsils which are infected and which are of potential danger as foci of infection.

Because of recurrent tonsillar infection, **chronic tonsillitis** with a **buried tonsil** may result. In this condition, the faucial pillars become inflamed and adhere to the surface of the tonsils, occluding the exits of many of the crypts. The adhesions may be so extensive that they obliterate the supratonsillar fossa. This absorption from the tonsils gives rise to **chronic cervical** adenitis and often to a low grade toxemia which interferes with nutrition to such a degree that the children are underweight and anemic. The adenitis of this form of tonsillitis is too often considered to be tuberculous and unnecessary operations have been performed for its relief. It is this type of tonsil which is often etiologic in nephritis and in *blood stream infections* with *arthritis* and *endocarditis.* Removal of buried tonsils is always in order, especially if the infant shows evidence of a toxemia. Most buried tonsils removed contain small abscesses.

As a result of nasopharyngitis or pharyngitis brought about by a diplo-streptococcus, the condition first described by Pfeiffer as **glandular fever** appears. The evidences of infection within the throat are slight and may escape notice. A little reddening, no tenderness, a moderate enlargement of the tonsils and adenoids, if these be present, constitute the pharyngeal and nasopharyngeal appearances. The most striking physical sign is the sudden enlargement of groups of cervical glands. A group of three or four glands becomes swollen, the swelling usually at-

tains an extreme degree within a few hours, and the affected group remains enlarged for some days. About the time these glands begin to diminish in size or perhaps a day or two before, a similar group in another part of the neck undergoes the same changes. There may even be a space of a day or two between the cessation of the signs in one group and the onset of swelling in another. Group after group is affected, and the disturbance runs a course of from 3 to 6 weeks. Pain is a marked feature in many of the cases, and even when there is no spontaneous pain, the glands are always tender to the touch. Sometimes the swelling is so extreme that it is a matter of difficulty to be certain that suppuration is not occurring in the deeper glands of the swollen group. In about 1 per cent of the cases suppuration does occur in one or two of the glands and when it does, evacuation through the smallest possible incision becomes necessary.

In Pfeiffer's original description, he laid stress upon enlargement of the spleen as a constant clinical expression of the disease. Cases of this malady have been common in our experience of late and in only about 10 per cent of them has splenic enlargement been pronounced.

The peculiar behavior of the temperature is an arresting feature of the condition. It is usual for the disease to herald its onset by a rigor even in rather young infants. This may occur 24 hours before the appearance of any cervical gland enlargement. Shortly after the rigor, the temperature may rise to an extreme height, even to 106° or 107°, although the usual maximum is about 105°. Within an hour or two, the thermometer may register not more than 100°. Occasionally, the registration may be subnormal. Throughout the course of the disease, these extreme variations are the rule—hyperpyrexia at one time of the day and a moderate or low degree of fever at another. It is never possible to foretell at what hour the rise will come or at what time of day the remission; 24 or 48 hours may pass during which the thermometer may register only moderate degrees of fever. Then with the enlargement of a new group of glands, another rigor may take place followed by a hyperpyrexial swing and another two or three days of varying fever.

The malaise is as variable as the fever and it is more or less dependent on the height of the temperature. The irritability of the child is striking; it seems to depend upon the develop-

ment of a mild acidosis which accompanies the infection. The urine is always highly acid; it may contain albumen, a few hyaline and granular casts and a few pus cells. It is frequently possible to culture, using blood agar, the diplo-streptococcus from the urine of these patients. The blood picture is of a moderate leucocytosis. The differential count is not informative.

Complications in this disease are rare. Endocarditis has occurred, nephritis also. The writers have seen a case of peritonitis and one of meningitis develop in the course of the disease. In both of these, the characteristic diplo-streptococcus was demonstrated as the etiologic factor of the complication.

The *treatment* consists of local applications to the swollen glands, hydrotherapy to control the pyrexia, cleansing applications to the mucous membrane of the nose and throat, the exhibition of alkalies and fluids in order to combat the threatening acidosis, and the maintenance of nutrition. Salicylates have been given, but apparently they have no effect on the morbid process.

The ice bag is preferred as a local application when the infants tolerate it. Hot applications are more apt to encourage suppuration and are to be avoided, although for those patients who do not endure cold well, warm compresses may be gratifying.

The nose and throat and nasopharynx are sprayed every 4 hours during the day with a dilute alkaline solution such as 2 per cent borax or dilute alkaline antiseptic solution.

For the higher degrees of fever, there is no measure so adequate as the tepid pack. (See Methods.) The weaned infant should be induced to take at least 1½ pints of fluid a day, and this may well be a 10 per cent lactose solution or orangeade. (See Recipes.) Citrate of potash (40 to 50 grains daily) may be administered. For the graver degrees of acidosis, it may be necessary to administer fluids intraperitoneally or intravenously. (See Methods.)

Milk is badly tolerated by most babies who suffer from glandular fever. In order to keep up the nutrition, lactose, dextrins, egg-yolk, fruit juices and fruit sauces should constitute the bulk of the diet. If there is any indication of a saccharolytic diarrhea, protein-milk should be the main reliance until the stools have improved. (See Recipes.) In the stage of convalescence, anemia must be overcome. For this purpose, the iron-containing foods such as egg-yolk, meat or liver pulp, spinach, arti-

choke and apple should be added to the dietary, and the saccharated carbonate of iron may be used in doses of 2 grains, four times a day. Should the anemia be extreme and malnutrition a feature, injections of the cacodylate of iron, 8 to 10 minims (of a 5 per cent solution) daily for 6 or 7 doses and then on alternate days for a like number, are in order.

Suppuration about the tonsil **(peritonsillar abscess, quinsy)** happens as a sequela to acute tonsillitis, but it is not frequent in infancy. The pus accumulates about the outside of the capsule of the tonsil and pushes its way into the areolar tissue of the soft palate. In the course of the suppuration, the tonsil is bulged inward into the lumen of the throat and lies there visibly swollen and red in close approximation to the swollen soft palate. The uvula is edematous and twisted out of its normal relation to the midline. When the abscess has been present for some days, fluctuation may be detected by palpation over the soft palate. If treatment be delayed, the abscess ruptures either at the upper pole of the tonsil or just above the supratonsillar fossa through the soft palate. The process of softening and erosion may include a blood vessel, and the spontaneous evacuation of pus may be accompanied by a good deal of hemorrhage. The infant with quinsy appears very ill, and is obviously suffering from pain. Dysphagia is a marked feature of the condition, and as a result of the disinclination to swallow, saliva and mucus accumulates in the mouth, or the child may drool the gathered secretions constantly out over the lips. The breath is offensive, the tongue is thickly coated, and the mouth is opened with difficulty. The symptomatology of **paratonsillar abscess** is almost the same but this suppurative affection is the result of the breaking down of one of the deep cervical glands that lies a close neighbor to the tonsil. In such a case, the palate is unlikely to be involved and the tonsil is pushed forward rather than inward by the abscess.

The *treatment* of these abscesses is by incision and evacuation of the pus.

The glands which lie in the retropharyngeal space undergo suppuration to form **retropharyngeal abscesses**. These abscesses are much more frequent in infancy than they are in later life and they are of more frequent occurrence than the paratonsillar abscesses. Attention is usually first drawn to this form by the obstruction to breathing. The earliest evidence is a snoring which progressively becomes more intense. Dyspnea supervenes and

the child suffers from attacks of threatened suffocation. The supine position intensifies the attacks and as an emergency measure, relief may sometimes be had by inverting the child. Dysphagia is a less marked symptom than dyspnea. It is of later onset and when it is present, it suggests that the abscess lies low down in the neck. Manual examination of the cervical region reveals swelling on one or both sides. If the child be restrained by wrapping it in a sheet, examination of the pharynx under a good light will show that the posterior wall is bulging forward on one side, usually the same side upon which the enlarged, tender, cervical glands lie. The fluctuation may be detected by palpation over the red protruding mass. There is no such rigidity of the neck with this form of retropharyngeal abscess as there is when spinal caries is the source of the suppuration. There is, however, tenderness and a certain degree of stiffness of the muscles due to myositis and to adenitis of the cervical lymph glands. Occasionally, an abscess of similar nature may appear high up in the midline of the nasopharynx, pointing between the pharyngeal aponeurosis and a mass of infected adenoids. Dysphagia, of course, is no symptom of such an abscess, but dyspnea, the result of extreme nasal obstruction, may appear. The diagnosis must rest upon evidence of fluctuation discovered by the finger palpating the nasopharynx.

The *treatment* of retropharyngeal abscess is essentially surgical. The position of the child at the moment that the pus is evacuated is important. He must be held so that the pus is prevented from entering the larynx. It is rarely necessary to use a knife to incise such an abscess. The point of a curved hemostat may be thrust through the abscess wall. Occasionally, there is a yellow spot of tumefaction which indicates the thinnest portion of the abscess wall. Once thoroughly evacuated, retropharyngeal abscesses are unlikely to recur.

A much rarer form of retropharyngeal bulging from pus production occurs when there is caries of the cervical vertebræ. Under such circumstances, the pus may gather in the space between the spinal column and the prevertebral fascia. These retropharyngeal abscesses of **spinal origin** show a clinical picture that differs from that of the ordinary retropharyngeal abscess. The patient is known to be suffering from tuberculosis of the cervical spine. The neck muscles are rigid, the head is carried care-

fully and moved as little as possible, the fever is slight or absent. The most striking symptoms are immobility, dyspnea and dysphagia.

The *treatment* is immediate evacuation to relieve the pressure of the encapsulated abscess. In this form of abscess, the incision should always be made in the neck in order that the pus may be evacuated externally. This is in contradistinction to the choice of site for incision in the ordinary acute, glandular, retropharyngeal abscess.

Among the less serious affections of the respiratory tract in infancy, there is none that gives a more alarming picture than **spasmodic laryngitis (croup).** In infant patients the first evidence of the approaching croup is a brassy cough. At first, this cough is occasional, and in the milder cases it may not be repeated with any great degree of frequency. There is some mild discomfort evident in the swallowing of saliva. In the more severe cases the brassy cough becomes constant and harassing.

Whenever an infant has such a laryngeal cough, secretion taken from about the glottis should be smeared for examination, and it should be cultured both on blood serum and on glucose agar. Such a test will be final in differentiating spasmodic laryngitis from laryngeal diphtheria, although it is rare that the clinical picture will be confused by a good observer. It is characteristic of simple catarrhal laryngeal cough that it is absent early in the day, that it begins toward evening, and that its intensity increases during the night. Cyanosis and dyspnea, except in unusual cases, is present only when the cough is most intense; that is, at night. Even without treatment, improvement comes in the early morning hours. In many cases the attack is self-limited; usually, it lasts not more than a day or two; rarely, it is continued three or more days. Agar cultures from the throat secretions will always show cocci—usually pneumococci, less often streptococci either in chains or paired. In a large proportion of cases, croup is a complication or a sequela of rhinitis or of an infectious tonsillitis. However, a removal of the tonsils does not always prevent future attacks of croup, because the bacteria have already found lodgment in the mucous membrane of the lower pharynx and the larynx where they lie dormant in a state of balanced immunity, to be aroused to heightened pathogenicity by some accident in the child's environment.

There is usually but a slight increase in the temperature which rarely rises over 101° or 102° F. Acute laryngitis may be a

premonitory symptom of measles, influenza, pneumonia or diphtheria in infants, and the possibility that any one of these diseases may develop must be kept in mind whenever an acute laryngitis of an infant arises for consideration. Certain severe cases of catarrhal laryngitis are further complicated by the factor of *spasm in the larynx*. It is this additional feature which gives rise to the alarming appearances of croup; most often it complicates the picture only at night. The child wakes up struggling for breath and develops a well marked inspiratory stridor, sometimes to so great a degree that the parents are certain that the infant's death from the obstruction is imminent. The spasm of the glottis is always self-limited and it passes away after some seconds or a minute or two, to recur two or three times in the next few hours. If the child does not again fall asleep, he becomes exceedingly restless and frightened before the onset of another spasmodic seizure. There are very few diseases which attack infants that bring more terror to the hearts of parents, and it is gratifying that the physician is able to tranquilize them with the information that children attacked in this way never die from this particular phenomenon.

The rationale of the *treatment* of acute laryngitis must look to decreasing spasm, to increasing the amount and decreasing the viscidity of secretions while favoring their expulsion, to soothing the cough, and to quieting the nervous symptoms. When the cough is severe and the dyspnea moderate or severe, with or without cyanosis, immersion in a mustard bath or the routine of the mustard pack should be the first step to be undertaken. Remembering that the cough and the distress increase as night advances, the patient must be kept in an even, warm temperature, with a strict avoidance of draughts. If the sickroom is maintained at a temperature of 63° to 65°, and ventilation is insured by an open window in an adjoining room, the cough will be much diminished. Cold compresses, or for that matter hot ones, to the throat tend to reduce spasm. Steam inhalation decreases glottic contractions and at the same time encourages the production of a more voluminous and less tenacious secretion.

However, no matter to what degree we may use measures of physical therapy, our chief dependence must be on drugs. Of these, none is more efficacious to prevent recurrence than iodized calcium in doses of ⅓ grain every ¼ hour until 10 doses have been given. The tablets may be crushed and given in sugar

water. For older infants a 1 grain tablet crushed and dissolved in sugar water should be given until 20 or 30 grains have been taken. Ipecac to effect immediate relief, in the form of the syrup, is also useful and has the advantage of being found in almost any household medicine chest. The dose is 15 to 40 minims for a 15-pound child.

Another drug of value in the spasmodic, dyspneic stage is apomorphin. Given in doses sufficient just to bring on emesis, it has the virtue of a later action that soothes the nervous system and induces sleep. Furthermore, its availability is great because it is usually to be found in the hypodermic case, an inevitable part of every doctor's equipment. The drug should be given by mouth in solution, in small doses. For a 15-pound baby, 1/1500 to 1/1200 of a grain every 20 minutes until from $\frac{1}{120}$ to $\frac{1}{60}$ grain has been taken. In the most severe cases, the use of opium (codein or heroin) together with atropin becomes imperative. A 15-pound infant will accept a single dose of $\frac{1}{60}$ grain codein and 1/1000 of atropin. If this is tolerated well and the child is awake and still distressed after 3 or 4 hours, the dose should be repeated once only, unless there is some urgent reason for a further employment of the drug.

An infant may get a **foreign body in the nose.** This body may be wedged in the nostrils, or it may pass the nostrils and fall into the larynx to become entangled there, or it may fall through the larynx into the bronchus, in which case it is apt to find its way into the large, straight right bronchus and remain lodged somewhere in the bronchial tree of that side. It seldom happens that a foreign body of any size passes into the left bronchus. Older infants, however, may aspirate partially chewed food, especially almonds or peanuts. Many small foreign bodies will be thus scattered over the bronchial mucous membrane, producing widespread irritation. As an evidence of this irritation, a spasmodic bronchitis with purulent inflammation of the mucous membrane may be set up. As a result, pseudoasthmatic attacks will recur for some weeks, even months, until the superficial suppuration along the respiratory mucosa has been sufficient to carry out all the minute foreign bodies that have been aspirated.

When the foreign body has lodged in the *nose,* it usually stops in the anterior portion between the inferior turbinate and the septum. As a result, the mucous membrane in this situation is swollen, tumid and red. After the mass has remained for some

time, it becomes embedded in granulations which ulcerate and give rise to an offensive, purulent, and sometimes bloody discharge. The discharge, pouring down onto the lip, may cause an eczema, and this one-sided discharge with eczema due to the presence of the foreign body must be differentiated from a similar state of affairs which occurs in the course of a subacute or chronic nasal diphtheria. The offensiveness of the discharge caused by the presence of a foreign body is one point that aids in making a diagnosis, for in membranous rhinitis the discharge is rarely very offensive.

The visual observation of the foreign body by an examination of the nose will identify the cause, and it is sometimes possible to see the impacted mass only after the mucous membrane has been thoroughly cocainized and adrenalinized. The *treatment* of the condition is removal of the body. If this be soft, it can be withdrawn by ordinary forceps, but commonly a child gets hard bodies, most often a shoe button or a bean, and an attempt to remove such masses with forceps is rarely successful and considerable serious trauma of the mucous membranes may result from the attempt. When it is determined that the mass is such an article, it is better that the child be given a general anesthetic. When it is not thought advisable to anesthetize the patient, the nose is treated with cocaine and adrenalin. A blunt hook should then be passed through the middle meatus of the nose until its curve is well behind the slippery, impacted body. The hook is then turned so that the mass is included within the curve, and a gentle traction is made in a direction downward and forward. Unless the mass has been lodged for a long time, a general anesthetic will be unnecessary, and if the child is well restrained by being wrapped in a sheet, the local anesthesia produced by the cocaine and adrenalin solution will suffice to make the removal possible.

When the foreign body has slipped down into the *larynx,* immediately upon its entrance, spasmodic closure of the vocal cords ensues and a violent spasm of coughing is set up. This, as a rule, will force the body out into the pharynx and in the usual course of events it is swallowed. However, on rare occasions, especially if the body is fairly large, it may become impacted between the vocal cords and completely obstruct the entrance of air into the lung. As a result, the patient becomes rapidly asphyxiated

and dies. A little milk aspirated into the larynx of a nursing infant may bring about the same sort of spasm and prove fatal.

If the body is smaller, it may lodge in the pouch between the true and false vocal cords, and after the first urgent asphyxia passes away, it may give no further symptoms except a little huskiness of the voice due to a moderate edema of the laryngeal mucous membrane, including that of the vocal cords. When the irritating body is small, it is coughed out and the symptoms immediately cease. If the foreign body is not of the sort to obstruct respiration, it may lie in the larynx until it breaks up. In infants the result will be an ulceration of the mucous membrane, and later possibly a narrowing due to the contraction of the scar following destruction of the superficial areas of the mucosa.

Direct inspection of the larynx is the only absolute guide to diagnosis. An approved laryngoscope should be passed by a competent laryngologist whenever continued symptoms of laryngeal obstruction follow a history of choking at a meal. The radiogram is of only relative value in determining the presence of a small foreign body in the larynx. If the child has aspirated a large foreign body causing symptoms of asphyxia which threaten its life, no time should be lost before performing an emergency tracheotomy.

In case the foreign body has passed the bronchial bifurcation and lodged in the bronchus, the radiogram or an observation through the fluoroscope is a much greater aid to diagnosis. Metallic bodies will show clearly on the plate, and while soft bodies are not opaque to the rays, they usually swell up and so completely obstruct air entry through the bronchus in which they lie that areas of collapse appear in the lung and compensatory emphysema of the other lung is evident. The clinical signs of these areas—collapse, lack of air, decrease in resonance, and limitation in movement—are to be demonstrated. These, together with dyspnea and coughing without apparent reason, may well cause the examiner to suspect the presence of a foreign body in a bronchus, especially when the history permits the inference that such a body could have been aspirated. From the larger bronchi, the offending mass may be removed by the aid of the bronchoscope. Some of the soft masses, such as lumps of bread crust, had better be left untouched. After a little while they soften, and frequently they are coughed up and swallowed. The

sequela is not always so fortunate. The putrefying mass of foreign material may, and often does, set up a localized abscess, and as a result bronchiectatic cavities develop or the wall of the bronchus may be perforated and a lung abscess or a gangrene of the lung ensue. Occasionally the perforation enters the pleural cavity and causes an empyema, and the writers have seen a case in which the foreign body, having entered the pleural cavity, caused a localized empyema which perforated the chest wall and was spontaneously evacuated.

For the *examination of the chest of an infant*, somewhat different technic is needed than when the adult chest is under examination. It is wise to have the child undressed and wrapped in a covering that opens at the back. The baby may then be taken up by the nurse or attendant who should hold the child on her shoulder in such a way that the infant does not see the physician. (See Methods.) Instead of beginning the examination, as is usually done in the case of an adult, by inspection, followed by percussion, auscultation should be the first diagnostic measure undertaken. The examiner can very often auscultate the entire back of the chest before the child is aware of a stranger's presence. Much evidence of the conditions within the chest can be acquired while the child is crying and the contrast between the sounds heard in quiet and in forced respiration be made. Crying is of no disadvantage, in fact it is a positive aid to the examiner when he is inspecting or palpating the chest. During percussion, care must be taken to strike the pleximeter finger only at the moment of the child's full inspiration, for an erroneous impression of flatness may arise if the stroke is made at the moment of expiration when the lung is but slightly expanded.

An *inspection of the chest* of a normal infant reveals that its contour differs to a marked degree from that of an adult. A baby's chest is rounder, lacking the peculiar oval cross-section characteristic of the grown-up. This structural peculiarity modifies the possibility of pulmonary expansion in the infant whose lungs move up and down readily but are definitely restrained in their lateral movements. The relatively large size of the heart as it lies in the chest in its high position in relation to the diaphragm must not be forgotten. The essentially weak movements of the costal muscles and the limitations of pulmonary expansion and contraction rob the infant's heart of much of the aid that the more forceful inspiration of the adult brings to the help of the

circulation. The suction effect of breathing on the venous circulation is limited in the baby, with the result that relatively minor degrees of pulmonary involvement embarrass the circulation and lead quickly to dilatation of the right heart.

Bronchitis frequently follows the appearance of an acute coryza or of a tonsillitis in a baby. Very often inflammations about the nasopharynx are overlooked and bronchitis is diagnosed when the conditions in the bronchial tubes are subsidiary. The peculiarity of the symptoms of bronchitis in infancy depends upon the fact that spasm is readily induced in the tubes and that the tubes themselves are narrow and therefore respiration is obstructed by relatively small accumulations of secretion in the later stages, and by the swelling of the mucous membrane in the earlier days of the disorder. Such an obstruction in the medium and smaller sized tubes leads to the production of little areas of collapse, a phenomenon which contributes another factor to the production of respiratory obstruction with a consequent deficiency in the oxygenation of the blood. It is this interference with oxygen exchange that causes cyanosis and dyspnea to be so marked in the case of infant bronchitics. In some cases the factor of spasm in the tubes is dominant and the result is a **spasmodic bronchitis.** It is frequently a matter of extreme difficulty to decide whether the symptoms found in an infant's chest indicate spasmodic bronchitis or a true asthma. In the earlier stages of any bronchitis before secretion in the bronchial tubes is abundant, engorgement and swelling of the mucous membranes is always accompanied by some spasm. At this time the symptoms of the disease will depend upon the degree of obstruction. The fever is usually slight, 100° to 101° F., although, because of the instability of their heat-regulating apparatus, young infants may develop a hyperpyrexia; or on the other hand if they are premature or congenitally weak they may suffer from a severe bronchitis which runs an apyrexial course. In a young infant the appearance of a bronchitis with severe symptoms (urgent *dyspnea* and *cyanosis*) without fever is always of grave prognostic significance.

The respiration is less hurried than one would expect. In the early stages and in mild cases, the rate may not be raised much above the normal. With increasing obstruction to air entry in the tubes, there may be a rise in the respiration rate to 35 or 40 in a minute, but the increase is rarely more than is congruous with the rise in temperature. If the obstruction is extreme

and widespread and has induced much scattered, patchy collapse of the lung, then a slow respiration comes into evidence, and when such a slowing occurs, the child's condition must cause the attendant grave anxiety. Under these circumstances the dyspnea becomes urgent, cyanosis becomes marked, forced expiration appreciable, and the alæ nasi fall in with every breath. Infants with bronchitis give little evidence of pain except when they cough; not always then, although in the early stages of the disease each cough may be accompanied by a little protesting cry. The cough of this early stage is unproductive and frequent, but its unpleasant features soon disappear to be followed by a looser cough that occurs less often and that seems to be unnoticed by the child.

The *physical signs* found over the chest in bronchitis are largely auscultatory. Percussion reveals little except in extreme cases with forced breathing, and in mild cases there are no alterations that the observer can discern through an inspection of the movement of the chest. During the early days of the affection, the stethoscope reveals characteristic, harsh, whistling râles which may be heard in both inspiration and expiration. In spasmodic bronchitis such râles are many in number and of wide distribution, so much so that the term "music-box" chest has been well applied to the phenomenon. After a few days these dry râles are replaced by ordinary moist, bubbling râles which are produced for the most part in the large tubes. The presence of this type of râles indicates improvement in the bronchitis, and coincidentally with their appearance it becomes obvious that the child is coughing up and swallowing a good deal of bronchial secretion. At the same time, cyanosis abates and dyspnea gives way.

When the bronchioles are involved in a bronchitis, the condition known as **capillary bronchitis** ensues. Under these circumstances the entry of air into the alveoli is greatly obstructed, and as a result cyanosis, dyspnea and forced breathing may become extreme. The interference with the full dilatation of the air-cells brings about a falling in of the supraclavicular and subcostal regions. Such a clinical picture appearing in an infant is of most serious import and demands the closest observation, for death from asphyxia may occur unexpectedly with dramatic suddenness. It is not only asphyxia that threatens, but the interference with respiration reacts upon the heart, and circulatory failure has to be feared. Even extreme bronchitis with

alarming symptoms is well borne by vigorous infants, but the outlook is very grave when the disease attacks the premature, the congenitally weak, and the rachitic. Ordinarily in the case of a vigorous infant, the attack runs to defervescence and clears up within a week or two although in the asthenic child who does not succumb, the course of the affection may endure throughout weeks or months; or it may apparently clear up for a few days and a condition of recurrent bronchitis become established to the child's detriment. Recurrent and chronic bronchitis frequently complicate rickets, and one or the other is a constant accompaniment of enlarged adenoids, hypertrophied tonsils and chronic nasopharyngitis.

Bronchopneumonia is a frequent complication or sequela of bronchitis during infancy. It is common to classify bronchopneumonias as primary and secondary. A pure bronchopneumonia of primary origin is rarely encountered in clinical experience. In measles and influenza, bronchitis and bronchopneumonia are so common that they can be considered almost as part of the disease rather than as complications. Rhinitis, pharyngitis or bronchitis may be considered as constituting the prodromal stages of the bronchopneumonia or they may be considered as independent stages of which the bronchopneumonia is the sequela. The only respiratory disorder in the nature of bronchopneumonia which appears suddenly and apart from any other disease is the so-called **lobular pneumonia** and this is in truth not a bronchopneumonia at all but a disease in many ways analogous to lobar pneumonia. The only real difference is that the consolidation of the lung occurs in scattered lobules and does not include all the lobules of a given lobe of the lung.

Postmortem findings are responsible for many diagnoses of bronchopneumonia, for the condition develops as a *terminal infection* in many diseases of infancy which have run a course quite apart from any previous respiratory involvement.

The name **catarrhal pneumonia** has been used to describe certain forms of bronchopneumonia. This nomenclature attempts to be descriptive of the pathologic findings in the lungs. There is a catarrhal condition in the bronchial tubes, their mucous membrane is swollen and soft, and their lumen contains masses of desquamated softened epithelium in which many bacteria, pneumococci, staphylococci, streptococci, and often other organisms are growing in abundance. The walls of the bronchi

and bronchioles are thickened and the peribronchial tissues and the septa of the lungs are invaded by microorganisms. This thickening and the inflammatory process interfere with circulation and bring about congestion. Dilatation of the bronchioles may occur, minute abscesses and tiny hemorrhages resulting. Together with these pathologic manifestations, the air-cells become involved in areas of patchy pneumonia. In reality what happens is that the epithelial lining of the alveoli is shed, accumulates and, together with purulent exudate, fills up these essential spaces. Here and there throughout the lungs small bronchi become occluded and areas of collapse are added to the pathologic picture. *Enlargement of the bronchial and mediastinal glands* is an inevitable part of a pronounced bronchopneumonia and probably contributes to the production of the irritative cough. It must not be forgotten that this adenitis may persist for weeks or months after the other evidences of a bronchopneumonia have disappeared. This fact renders it necessary to exercise great care before deciding that a radiographic plate which shows shadows of enlarged glands represents a true tuberculosis of these structures. The essential difference between the pathologic condition in the lung of a child with lobar pneumonia and one with bronchopneumonia lies in the fact that the pathologic conditions in the latter follow a local infection of the pulmonary tissues, while the lung changes of the former represent a pulmonary immunity reaction in the course of a generalized infection by the pneumococcus.

The symptoms that reveal the presence of a bronchopneumonia are continuations or exaggerations of those found in the severer forms of bronchitis. After a few days of preliminary bronchitis or in the course of one of the infectious diseases, a sudden unexpected rise in the temperature occurs. It is not unusual for the fever to reach 105° or 106° F. at this time. The appearance of the pyrexia is accompanied by evidences of respiratory distress, dyspnea and prostration. The breathing seen in bronchopneumonia is characterized by an alteration in the normal respiratory rhythm. Ordinary inspiration is followed immediately by expiration, and on the completion of expiration there is a pause of from 1 to 2 seconds. When bronchial obstructive breathing is taking place, the pause occurs immediately after inspiration. A prolonged expiratory sound terminating in a grunt expressive of forced expiration completes the respiratory cycle. Ordinarily

in this disease, the rate of breathing does not exceed 50 respirations a minute, although it is not infrequent to find children suffering with bronchopneumonia whose respirations are 60, 80 or even 100 per minute. The higher rates must always be causes for alarm.

The fever, the diminished oxygenation of the blood, and the effects of circulatory embarrassment combine to raise the pulse rate which is always high in this disease. Sometimes it is so rapid as to be almost uncountable. However, the consideration of the pulse rate is of little value because the instability of the pulse during infancy renders alteration in its rate often misleading.

Patients with bronchopneumonia always *cough*. The character and intensity of the seizures will vary with the condition of the child, the extent of the involvement, and the stage of the disease. With a nervous or weakly infant, especially during the earlier days of the attack, the cough will be dry, unproductive and exhausting. Later, as improvement sets in, it becomes less constant and at the same time it is obvious that the child is raising secretion and swallowing it. A progressive weakening of the cough at a time when other symptoms are increasing in intensity is a sign of bad prognostic omen, for it indicates that the child's strength is becoming exhausted. Babies with a mild bronchopneumonia cry a great deal; they are especially apt to cry after they have coughed. Those who are suffering from attacks of a severer nature cry little; they are restless and fully occupied with the business of breathing. Crying but adds to their respiratory embarrassment and it is avoided to a remarkable degree. In infants affected in this way, the expiratory grunt typical of embarrassed breathing takes on the character of a moan. These severely ill children sleep but little. The fitful naps they take are constantly broken and they awake with a start unrefreshed. One of the causes contributing to a fatal ending in this affection is lack of sleep. The infants are thirsty and take water avidly, although for the most part they refuse food. The hot, dry skin from which most of them suffer is an added source of discomfort, although in the more severe cases, vasomotor disturbances may ensue and be followed by profuse perspiration. The replacement of cyanosis by a grayish pallor and the breaking out of a cold clammy sweat over the infant's skin is sometimes an agonal phenomenon.

The symptoms of the bronchopneumonia are so revealing that

physical signs do little more than confirm the diagnosis and indicate such complications as arise in the course of the disease. Inspection shows an overexpanded chest due to the excessive efforts of the respiratory muscle, and inspiratory dilatation of the nostrils together with a forced descent of the larynx at the same time. In cases where there is extensive involvement of the lungs, the subclavicular and subcostal areas of the chest may be observed to fall in with each respiration. Sounds heard through the stethoscope will vary with each chest examined and in the different parts even of one chest. The most striking feature of the auscultatory signs of bronchopneumonia is their great variability. At one point they may vary according to the character of the breathing, and before and after coughing the sounds differ. The passing of a large mass of mucus from a bronchus may bring about absolute alteration in the sounds heard over the lung below the point at which it had been lodged.

There are some chests in which other physical signs and symptoms permit no deduction except that extensive areas of consolidation are present in the lung; and yet auscultation will reveal nothing but crepitant râles. Even with the most severe cases of bronchopneumonia, such crepitant râles may be the only phenomena to be heard at any time in the course of the disease. More often, however, together with this type of râle, scattered patches of bronchial breathing are discernible and there will be isolated areas of silence suggesting the presence of collapse. When bronchial breathing is obvious, it may be of amphoric quality, accompanied by many coarse râles, and a combination of physical signs sufficiently like the physical signs of cavitation, may deceive even the best observer. The fact that over such an area there may be a percussion note analogous to the "cracked pot" note (considered to be pathognomonic of the presence of a cavity) does not diminish the possible confusion. Because of this possibility of error, diagnosis of a cavity in the lung of a young infant should be made with hesitation and be accepted as definite only after radiographic confirmation. There is little to be learned by percussion of the chest in the course of a bronchopneumonia. When there are large areas of collapse, patchy dullness may be encountered. More often there is a "boxy," hyperresonant sort of note. The pleura is much less frequently involved in this form of pneumonia than it is in the lobar form of the disease. Occasionally a few dry râles are heard in the course of a broncho-

pneumonia; infrequently an empyema occurs; and on very rare occasions pleural effusion of the serous type forms as a complication or sequela of the disorder.

Embarrassment of the heart and of the circulation can be considered more as part of the disease than as a complication of it. Congestion of the pulmonary vessels reacts on the heart. Almost always the right heart undergoes a dilatation, and it is not unusual for the cardiac embarrassment to be such that the liver shows progressive enlargement. The myocardium suffers not only from mechanical stress but from the toxic influence of bacterial poisons as well. The result of this myocarditis is seen in increasing tenuousness, weakness and rapidity of the pulse. Acute dilatation of the heart may occur during bronchopneumonia even when the child is vigorous, but it is much more apt to happen with the asthenic and rachitic. When a child undergoes such sudden cardiac failure, it may become pale, cease to breathe, and die within a few moments; or when the dilatation is more progressive, the clinical picture presented may be one of cyanosis with grouped respiration, intermittent dyspnea and bradycardia. These attacks are very alarming in appearance but not so fatal as the more acute seizures of cardiac dilatation with pallor.

Cyanosis often anticipates increase in fever when the child may become lethargic and even comatose. In the more severe cases convulsions come on after a few hours, the temperature shows still greater rise, sometimes 107° or 108° F. and the child dies. There are on the other hand, cases of severe bronchopneumonia which run their entire course, usually to a fatal ending, while the temperature remains subnormal. Fortunately these manifestations are not so frequent as those of a less profound nature. When the process in the lung is only moderately extensive, the cyanosis and dyspnea clear up after a few days, the fever diminishes in intensity and a long period of defervescence sets in during which the clinical picture becomes one of a severe or mild bronchitis rather than of bronchopneumonia.

Under favorable circumstances, the course of the disease is from 10 to 20 days, although in weakly or susceptible infants a relapse may occur in the midst of a period of defervescence to be followed after some days by improvement. This cycle of improvement and relapse may be repeated a number of times in succession. When this sort of relapsing bronchopneumonia comes on, an illness lasting 2 or 3 months may be the result. Such a clinical mani-

festation may terminate in recovery, but more often the patient becomes exhausted; each recurrence is borne with less resistance, and the child finally succumbs. Even when recovery from the respiratory disease does take place, the child, left weak and ailing, falls a ready victim to other infections especially those of the digestive tract. Not only as a termination but as a complication, diarrhea and malnutrition are encountered in the course of a recurrent bronchopneumonia.

The character of the room atmosphere is a matter of much moment in the *treatment* of a bronchitic. Physicians, moved by the successful use of cold fresh air as a therapeutic agent in adults, have recommended the same treatment for infants suffering from bronchitis and bronchopneumonia. Fresh air certainly is essential to the patient's well being, but the value of cold air is open to question. It has one great disadvantage—it aggravates cough, especially the earlier unproductive cough which may be so exhausting to the infant; therefore, the sickroom should be heated. A uniformly maintained room temperature of 60° to 63° F. is advantageous in the treatment of bronchitis. The warm room must, however, be well ventilated with windows open. When fog, wind or extremely cold weather prevail, it may be best to have the window of an adjoining room open to the outside and to secure ventilation by opening the doors between this room and the patient's chamber. Open fires have a reputation as efficient aids to ventilation, and they probably are, but it must not be forgotten that they are dusty and that the inhalation of dust may greatly aggravate a bronchial affection.

Good nursing is an essential in the treatment of bronchopneumonia. The intelligent fulfillment of the physician's orders in detail is often the decisive factor. Early report of the development of serious or suggestive symptoms may make it possible to institute measures for relief in time to prevent serious complications. To the nurse, the physician must look for assurance that the child is properly clothed. It is important that the patient be protected from chill and yet that he be not overclad and allowed to become soaked in his own perspiration. Careful nursing insures that bed covers do not weigh down on the already overburdened chest of the infant, and that poultices, bands and outgrown clothing do not further embarrass the struggling muscles of respiration. A bed cradle over which the bed covers are suspended is a real aid. Such a device carries the weight of the

covers and allows the chest free play. The child should be clothed lightly but completely. The rationale of the pneumonia jacket is difficult to compass except for those premature infants whose bronchial affection runs an afebrile course. To swathe a child who has a high ranging fever in a thick insulating jacket seems an absurdity, especially as the indications are for temperature reduction. Heavy poultices and tight clothing applied to the chest embarrass breathing; so does the all too popular abdominal band. These binders should be discarded as soon as the umbilical stump has fallen. They serve no useful purpose, they are often sources of discomfort, and tightly applied to the abdomen of a child who has an acute respiratory disease, they may be dangerous because of their interference with free respiration. The nurse should move the patient from one position to another as her discretion directs, but nothing should be allowed to interfere with the insurance of adequate rest for the child. It is just in this connection that good nursing proves its value. A tranquil, sensible woman will secure sleep and rest to a dyspneic, anxious child when an inexperienced, excitable nurse might only aggravate its disquiet.

Moistening the air to be inhaled seems definitely to help allay bronchial spasm and to loosen secretion. To provide steam, the croup kettle is an excellent device. (See Methods.) For home use, the model known as the Holt Croup Kettle is justly popular. Plain, unmedicated steam is a very effective inhalation, but many physicians prefer to medicate it by the addition of balsams to the water in the kettle. Compound tincture of benzoin, 1 dram, oil of eucalyptus, 1 dram, and turpentine, 15 or 20 drops, to a pint of water all are highly recommended drugs for this purpose. For hospital use, one of the electrically heated kettles is used. This form of kettle is absolutely safe. With the type of kettle used in household practice there is a naked flame, and with a naked flame there is always the possibility of an accidental blaze. Failing a proper croup kettle, a perfectly satisfactory substitute can be fabricated from an old tea-kettle with a piece of rubber hose slipped over the spout. To give steam inhalations continuously is a bad practice. A canopy open at least at one end and one side should be arranged in order to concentrate the moisture near the child, and the steam be led under this canopy should be used not more than ½ an hour out of each 2 or 3 hours. Closed tents, especially if the patient is placed in them overclothed, tend

to produce inordinate sweating, a result that aggravates rather than soothes the symptoms of respiratory distress. Early in the course of the disease, steam inhalations are of great value, for they undoubtedly contribute to loosening secretions and diminishing spasm. Later, when secretion is free and the infant is bringing up and swallowing sputum easily, there is no result to be expected from the use of such a measure.

In the early stages of bronchial affections when secretion is scanty and bronchial spasm present, hydrotherapy is of paramount usefulness. For mild cases, plain tepid or hot packs are indicated. When bronchitis is moderate or bronchopneumonia is mild, the mustard bath may be given with the certainty that it will bring relief, while for severe cases with bronchitic or bronchopneumonic symptoms, the application of the mustard pack is often a life-saving measure. It is especially indicated when there is cyanosis or any other indication of circulatory embarrassment in the course of a bronchopneumonia, or if toxemia is profound as shown by torpor, drowsiness or apathy. This form of pack is furthermore one of the most effective antipyretic measures at our command. (For details of these hydrotherapeutic measures see Methods.) When cough and thoracic pain are dominant symptoms, the application of mustard cloths or light flour and mustard poultices may be useful. The cloths are prepared by wringing out large pieces of flannel from a bowl containing 2 quarts of hot water into which 3 tablespoonfuls of mustard have been stirred. It is necessary always to make a paste of the mustard by stirring it up with cold water before it is added to the hot water, otherwise the thermolabile enzyme which releases the effective oil is destroyed by heat before it can become effective. To prepare the mustard poultices, the mustard is made into a paste with cold water and added in the proportion of 1 part to 6 parts of flour. The mixture is spread very thin and applied. Such preparations should always be applied to the back and sides of the chest; never should they be put on in front to bear down on the ribs. Instead of mustard cloths, turpentine stupes may be used. These are prepared by wringing flannel out of a mixture of 2 teaspoonfuls of turpentine to a quart of hot water.

Few available measures are more effective in encouraging expectoration than the ingestion of hot liquids. For this reason, it is well during the earlier days of the disease to restrict the

intake largely to fluids given in small quantities, as hot as can be borne and at short intervals. Hot skimmed milk with 1 grain of soda bicarbonate to the ounce given every 15 minutes to ½ hour in 2-teaspoonful to 2-tablespoonful doses may render the use of other remedies unnecessary. Other fluids may be used in a like manner; among those useful are broths, cereal decoctions, high calorie orangeade and flaxseed orangeade. (See Recipes.)

Medication is of distinct value as an adjunct to hydrotherapeutic and dietetic measures in the treatment of bronchial diseases. For spasm when that is dominant, an immediate injection of atropin, $\frac{1}{300}$ grain, adrenalin chloride solution 1 to 3500, 8 drops to a child of 2 years weighing 27 to 30 pounds, will almost certainly overcome the constriction in the tubes. The injection may be repeated once or twice at 24 hour intervals, but usually a single injection suffices. The use of the iodides is attended by a prompt increase in the amount of the bronchial secretion and by a decrease in its viscidity. A formula well suited for the dry stage of bronchitis is made up by putting 20 drops of syrup of hydriodic acid, 1 drop spirits of chloroform, and 1 grain sodium bromide in a teaspoonful of water and giving it every 2 hours for 4 or 6 doses, and thereafter every 4 hours for 1 or 2 days. This dosage is appropriate for a child 1 year old weighing 23 pounds. Once the secretion is well established, it is well to omit all drugs. If there be any symptoms of impending acidosis, these should be combated by the free use of fluids by mouth or if necessary by rectum, peritoneum, or vein.

When in the early stage of a bronchial affection cough is unproductive and disturbing to the patient, opiates may be used in an attempt to lessen it. Unless the cough is so incessant that it threatens exhaustion, the opiate had best be reserved for use in a single dose to be given in the early evening to be once repeated during the night at the physicians discretion. Codein $\frac{1}{16}$ grain, or heroin $\frac{1}{40}$ grain, may be combined in a mixture with sodium bromide 2 grains, glycerin 15 minims, and chloroform water enough to make a teaspoonful. This dose is appropriate for a 25 pound infant of 1 year. Tincture of lobelia has a well-earned reputation in the treatment of subacute bronchitis in which spasm is a factor of importance. Three minim doses every 4 hours for a few days may be useful. It is best combined with syrup of hydriodic acid, 15 minims, added to enough syrup of wild cherry and chloroform water to make a teaspoonful.

When acute *dilatation of the heart* is in evidence, intravenous injection of $\frac{1}{600}$ grain of strophanthin is often a prompt and effective remedy. This single injection may be followed by the use of 2 to 5 minims of tincture of digitalis by mouth every 4 or 6 hours until the heart muscle shows improvement evidenced by a slower more powerful pulse. On the whole, except in very severe bronchopneumonias, heart stimulants will not be needed. When the dilatation of the right heart is such that cardiac failure seems imminent venesection is indicated. The withdrawal of 3 or 4 ounces of blood from a 25 pound child is well borne and may prove to be a life-saving measure.

In bronchopneumonia, *true respiratory failure* may threaten life. The symptoms, dyspnea, cyanosis and restlessness, under these circumstances are independent of the extent of pulmonary involvement and they may appear while the heart is acting with power. They seem to be indices of damage of the medulla by absorbed toxins. The mustard pack is most effective in the treatment of this complication. A single dose of atropin $\frac{1}{300}$ grain, may be given while the pack is being prepared. Oxygen will be of service if the child is allowed to inhale it while lying in the mustard pack. Alcohol as simple elixir or whiskey or brandy in 15 to 20 drop doses every three hours quiets the nervous system and often produces sleep when other measures fail. Stimulants may be necessary when there is evidence of circulatory embarrassment. The best heart stimulant is the mustard pack. This measure may be re-enforced by the use of injections of atropin $\frac{1}{600}$ grain, camphor 10 per cent in olive oil, 10 minims, every 3 hours. Camphor in oil may be given as often as every $\frac{1}{2}$ hour when the pulse is failing progressively.

In the stage of defervescence and during *convalescence* from pulmonary affections, no better drug than cod-liver oil can be found. Combined in a palatable emulsion with the syrup of ferrous iodide in generous doses, this fat acts promptly and efficiently as a restorative. If anemia is profound and metabolism is affected, then injections of iron cacodylate will be useful. Seven minims of a 5 per cent solution every day for 10 days, then every second day for 10 more days, will be the dosage useful for a 25-pound child.

Almost invariably in the course of bronchopneumonia the *digestion* is disturbed, abdominal distention follows, and often there is a complicating diarrhea. Under these circumstances, a single dose of castor oil, 3 drams to a child 2 years old, will cause an

evacuation of the bowels. If distention is great, ⅓ c. c. of pituitrin injected subcutaneously usually will effect a prompt flattening of the abdomen. The diarrhea responds to an appropriate diet. If it be due to a carbohydrate overfeeding, with green, sour, excoriating stools, protein-milk is the feeding of choice. On the other hand, when the stools are foul, with brown mucus containing water, then malt-soup will usually effect a prompt amelioration of the bowel state.

The mouth and nose must be cleansed with scrupulous care. Syringing of the nose (see Methods) should be carried out daily. Scrutiny of the ears is essential, and a paracentesis should be promptly done if need be. Empyema, while a rare complication of bronchopneumonia, does occur; therefore, in every case the chest should be watched for the earliest signs of purulent effusion.

It is essential that the deadliness of bronchopneumonia, especially as it attacks the *premature and weak infant,* be kept in mind and every effort be made to prevent the incidence and spread of respiratory disorders. The premature and congenitally weak should be protected from cold, but also they must be given ample air and their living rooms must not be overheated. None who are subject to upper respiratory tract infection should be allowed access to the weakly infant. None should be allowed to kiss the baby or to touch the infant's head, face or lips with unwashed hands, neither should they be allowed to play with and fondle the baby's hands which may in a few moments be in the child's mouth. Hand to hand infection is now recognized as a very frequent source of infection; therefore, every effort should be taken to prevent the spread of disease to the infant in this way.

Certain children are subject to true asthmatic attacks. The earliest evidences of the **asthma** may appear within the first six months of life, although usually they are delayed until after the sixth or eighth month. The appearance of an asthma in an infant may indicate that the child is the member of a family in which asthma appears from generation to generation. There is a curious coincidence of infantile eczema and asthma. Oftentimes the asthma appears in the child who has previously been affected with an extensive eczema, or on the other hand, different children in the same family may be affected, one with the pulmonary and the other with the skin manifestation. There is no doubt that the present view that many cases of asthma are expressions of an anaphylaxis is true, although the number of instances in which

the *sensitizing protein* is discoverable with certainty is not great. Skin reactions to the proteins of certain foods may be discovered, and it will be found that in some of these cases, ingestion of this food initiates an attack of asthma. Another class of proteins which sensitizes and is responsible not only for asthmas but for rhinorrheas as well, includes the pollens. Probably certain bacterial proteins may also act in a like manner. It is conceivable that a child whose protective mechanism is readily unbalanced and who goes into anaphylaxis with ease may be the subject of invasion by bacteria. These bacteria passing the bronchial epithelium to lodge in the peribronchial tissues may acquire a state of balanced immunity and remain quiescent, producing no symptoms. A little later other like or related bacteria are inhaled, destroyed and their proteins absorbed into the blood stream. Such an absorption is in effect equivalent to the injection of a foreign protein to which the body has become sensitive. It is a well-known fact that the injection of tuberculin into an individual with a focus of latent tuberculosis overcomes latency and arouses activity in the infected area. It seems quite possible that the protein of dead bacterial bodies absorbed from the nose or other parts of the upper respiratory tract circulating in the blood may stir up the living bacteria lying about the bronchi and be responsible for the development of that type of anaphylactic shock with bronchial spasm which we call asthma. Undoubtedly other irritants besides proteins may contribute to the beginning of a bronchial spasm and produce a state of affairs very like a true bronchitis. This is seen following insufflations of powders, accidents which sometimes happen during the first few years of life. Mechanical irritation from the presence of adenoids and the pressure of enlarged bronchial glands have also to be remembered in their rôle as possible initiators of attacks of bronchial spasm indistinguishable from true asthmatic seizures.

The relationship of the activities of the glands of internal secretion to anaphylaxis is being studied with a great deal of care and some interesting facts are coming to light. It seems to be clearly proved that the adrenal is closely influential in the production of this group of symptoms and in some cases there is clinical evidence that the thyroid gland as well plays a part. Certain cases of asthma have responded to thyroid extract fed in appropriate doses, and the immediate and very striking though transitory relief which follows the injection of adrenalin into a patient suffering

from the respiratory obstruction of asthma is an acknowledged clinical fact, a fact that has proved of great therapeutic utility.

The most striking thing about an asthmatic attack is the abruptness of its onset and the rapidity with which overexpansion of the chest and dyspnea develop, although a close inquiry of the hours preceding the attack will show that the infant has been restless, perhaps showing an undue degree of excitability. In young infants an incessant hacking cough is often a feature, but the chief symptoms are those of obstruction to air entry with marked restlessness and great distress. Fever is usually wanting and the signs of cardiac involvement are much less than would be expected from the amount of repiratory embarrassment. At the height of the attack some cyanosis is present; it may be extreme but is usually slight.

Inspection of the chest reveals hyperdistention. The cage is fixed in a position of partial inspiration and the breathing is much shallower and more rapid than it should be. Percussion gives a hyperresonant note. It is particularly during an asthmatic attack that the distention of the air-cells becomes great enough to obliterate the area of cardiac dullness. Auscultation reveals everywhere over the chest a characteristic so-called "music-box" râle accompanying a respiratory rhythm in which the inspiration and expiration are both prolonged in time. The râles are dry and of variable size because they are produced both in medium and in minor tubes. After the attack has lasted a day or two, little by little the dry râles are replaced by moist ones and the signs of a mild, medium tube bronchitis ensue. It is a curious fact that the sounds heard in the chest are not always comparable to the amount of the apparent respiratory obstruction. The orthopnea, forced breathing and overdistention of the chest may pass away within a few hours, or after a day or two while the auscultatory signs are still at their height. During an asthmatic attack, especially when it affects a young infant several of these orthopneic seizures may come and go; each may last anywhere from 1 to 5 or 6 hours and the whole attack may persist over 5 or 6 days or even a week. Recurrence of the attacks is the rule. Their frequency will vary with the individual infant, with its sensitivity, and with the care with which it is guarded from all sources of mechanical and bacterial irritation.

Certain infants seem particularly susceptible to the inhalation of cold air, and attacks of asthma and of recurrent bronchitis may

often be prevented by insuring that while their sleeping rooms are well ventilated, the temperature does not drop below 62° or 63° F.

Great care must be exercised to prevent asthmatic infants from acquiring respiratory infections; therefore, all adults with colds or any other form of nose or throat trouble should be rigidly excluded from close contact with them. Especial precaution should be taken that no one should kiss the face or hands of such children, for a child's hands almost inevitably find their way into its mouth, and hand contamination is a frequent source of infection for little babies. A mother subject to an upper respiratory tract infection should be instructed always to wear a folded gauze veil during nursing periods in order that she may not subject her baby to droplet infection. Older infants who show the evidences of adenoid or tonsillar infection should be given the benefit of early removal of the offending structures, although it cannot be expected that the removal of adenoids will cure the asthmatic condition. Fresh air, sunlight and an appropriate dietary are essentials in any attempt to prevent the recurrence of the attacks.

As already noted, skin tests often show asthmatic infants to be sensitive to egg-albumen and sometimes to the protein of bovine milk. Most rarely a skin reaction will appear after the application of human milk. In the presence of these reactions, the offending food should be withheld while the child is young, and later, after its first birthday, an attempt can be made to immunize it by beginning to feed it minute amounts of the protein in question. By beginning with $\frac{1}{1000}$ of a milligram of egg white in solution and increasing the dose very gradually daily through 2 or 3 months almost any egg sensitive child can be given an immunity. Milk-proteins fed in the same way will sometimes immunize an infant. If attempts at immunization are delayed till later childhood they are rarely successful. Those infants who are sensitive to cow's milk are often able to take goat's milk without trouble, but on the whole if such a child is encountered in practice, it is much better to employ a wet-nurse until the child is old enough to accept a general diet. Older children may be sensitive to foods other than egg and milk. Almost any protein, including cereal protein, may react badly. Fortunately it is rare for a child to be sensitive to more than one protein; this renders it easy to prepare a diet which excludes the offending substance if it can be discovered. Aside from eggs and milk, orange, chocolate, barley and rice carry the proteins

which seem most often to produce asthmas, eczemas and urticarias.

In the presence of an attack, an immediate injection of 1/400 grain of atropin with 8 minims of a 1/3500 solution of adrenalin chloride for a 1 year infant will often check a seizure as if by magic. It is often unnecessary to repeat the injection, but in case the effect is either not immediate or is only transitory, one or two repetitions of the injection may be made at 4 or 6 hour intervals. The earlier the injection is given after the onset of the attack, the more likely it is to be effective, although there is a proportion of cases in which the use of this remedy is followed by no result. For such patients, relief often may be obtained by using a spray of dionin in 2 per cent solution. The two measures may be taken together when cases are very severe, although the spray should never be prescribed for home use but should be used by the physician himself or for him by a trusted nurse.

In order to hasten the production of secretions and the termination of the attack, there is no remedy equal to the iodides given in large doses. The addition of spirits of chloroform tends to aid in allaying spasm of the tubes. A formula which calls for 1 minim of spirits of chloroform, 20 drops of syrup hydriodic acid, 3 grains of bromide of soda in equal parts of syrup of raspberry and water, may be given a 25 pound child every 2 hours for 20 to 24 doses. The result of this is usually gratifying; the spasm and dyspnea disappear promptly, the cough becomes less irritating and more productive and the child brings up secretions freely and swallows them. In the intervals between attacks, the iodides are also valuable and apparently contribute something to diminishing the frequency of the seizures and minimizing their intensity. For long-continued use, the syrup of iodide of iron seems the best form in which to give the iodide. It can be given in from 5 to 10 drop doses diluted in water or if the child is rachitic or suffers from malnutrition, it may be combined with a 33 per cent emulsion of cod-liver oil.

On the assumption that asthmatic attacks are anaphylactic in nature, autogenous vaccines have been used with the hope that their injection would desensitize the asthmatic patient. Most often the vaccines have been prepared from cultures of bacteria recovered from the sputum at the height of the asthmatic seizure; such vaccines are often of definite value in reducing the number of attacks and in diminishing their severity.

Following the suggestion of Danysz, the writers have also used vaccines prepared from cultures of the bacteria recovered from the patient's stools. The use of the Danysz vaccines has been attended with more success than the use of simple autogenous vaccines of respiratory origin. It is probable that the amelioration of symptoms is not due to any specific bacterial inhibition, but is an example of that little understood phenomenon, nonspecific protein desensitization. This probability is emphasized by the fact that vaccines prepared by the method of Ross and Hutchinson (in which additions of guinea pig serum are made to the finished vaccine) are definitely more effective than those vaccines prepared from the same bacterial strains, but without this foreign protein addition. It is usual to begin vaccine treatment during a period of quiescence with an injection of 50,000,000 bacteria. The dose is repeated, but doubled each day for 5 days. In six weeks a second similar course is given and a third, 3 months later.

If the influence of the impaired activity of thyroid, pituitary, or adrenal gland is potent in producing anaphylactic phenomena, and if these are truly the cause of asthmatic seizures, gland therapy is a rational procedure; it is true that some asthmatic children have seemed to gain relief through glandular therapy. The use of small doses of whole pituitary, or of thyroid gland either alone or together with vaccine therapy whenever there are evidences of even slight dysfunction of these glands seems warranted by the facts experience has gathered.

The fact that asthmatics are free from attack in one climate while they suffer in another is well attested in medical experience, and it may be necessary to remove a child from one region to another in order to give it relief. However, it is rarely an important contribution to the treatment. Hardly ever will it be necessary for a medical man to impose great sacrifice on a family in order that an asthmatic child may have a change of climate.

There is a belief that **lobar pneumonia** is uncommon during the first two years of life. This belief is not warranted by clinical experience. The lobular and lobar forms of pneumonia, (which should be classed together as a distinct entity) are quite as common as bronchopneumonia. It is not demonstrable but it is probable that the lung signs in this affection are simply an expression of the efforts of pulmonary epithelium to establish an immunity against pneumococci. It is a well-known fact that the alveolar epithelium of certain animals used in the laboratory to study in-

fectious diseases has specific immunizing powers. This is especially true of the guinea pig whose pulmonary epithelial reactions to the typhoid bacillus have been studied extensively by Karl Meyer. By analogy then it is permissible, at least to consider the possibilities that the pneumonic process in the lung of the human subject, especially of the human infant, is of a similar nature.

That the disease has been considered to be rare in the early years of life results from the fact that the clear-cut physical signs of the lung involvement often fail in the earlier days of the disease. Indeed it is a common clinical experience to meet an infant patient who runs a typical symptomatic course and in whom the signs of extensive consolidation are delayed until after a crisis. In older patients, such misleading findings are attributed to a peripheral distribution of the consolidation; it is well known that unless the consolidation proceeds deeply enough to involve bronchi of fair size, tubular breathing is not heard. Furthermore, in case of infants who breathe so shallowly and so rapidly under the stimulus of minor degrees of fever and who cry and struggle so readily under examination, the determination of the signs of lobar pneumonia may be no easy task.

A carefully taken history, a prolonged scrutiny of the child as it lies or sits quietly undisturbed, and a painstaking examination of the lungs may be necessary before a right decision can be arrived at when lobar pneumonia is suspected in an infant patient. The history is helpful. The physician is frequently not called to see the child until after it has been ill for some days; but if the fact can be elicited that the disease began abruptly, this evidence may be of aid in arriving at a diagnosis. In very young infants, this sudden onset may be ushered in with a convulsion or by an attack of rapidly oncoming cyanosis with torpor. Often the first evidences of disturbance are vomiting (which is usually not prolonged) and diarrhea (which may amount to the evacuation of but two or three movements or which may be so profound and persistent as to divert attention entirely from the pulmonary features of the case). In older infants who are able to make complaint, the same symptoms may be present and the child may also complain of feeling cold and of pain in the head.

One of the most perplexing forms of onset is that in which the central nervous system is extensively affected. Some writers are so impressed with this phenomenon that they have classified it under the term "**cerebral pneumonia.**" These cerebral symptoms

show wide variation. When they are most severe, there may be perfect mimicry of meningitis. However, in all cases of lobar pneumonia in the very young the dominance of nervous symptoms is striking. Vomiting and delirium are frequent. In the so-called cerebral type, the disease usually begins with a convulsion, although this is not true in every case, for very profound involvement of the nervous system may take place without a convulsion. The child drops into a state of drowsiness which may deepen into a lethargy or into a profound stupor broken by periods of excitement and delirium. Tremor is often marked. The delirium is evidenced by attacks of screaming and great restlessness among the very young and by a wild babbling among the older infants. In this complication of pneumonia, head retraction is often an early and pronounced physical sign and in many of the patients, muscular rigidity is so extreme that it is difficult to believe that a true meningitis has not developed. As pneumococcus meningitis does happen with extreme rarity in the course of lobar pneumonia, this fact contributes much to the perplexity of the examiner and renders a lumbar puncture the only means of knowing just what is happening in the cerebrospinal system. It must never be forgotten that the same group of symptoms may indicate an involvement of the middle ear which is one of the commoner accompaniments of respiratory disorders during infancy. Lumbar puncture (see Methods) in an ordinary cerebral pneumonia will reveal if the child is suffering from what French physicians have called **meningism** which is apparently a sterile form of mild meningitis perhaps accompanied by a transient coincident encephalitis. The spinal fluid withdrawn in the presence of a meningism is always under pressure and relief of this pressure is followed by prompt amelioration of the cerebral symptoms. On examination, the fluid is found to contain few cells, no bacteria and very small amounts of globulin. Even without lumbar puncture, the cerebral symptoms disappear coincidentally with the appearance of the crisis. On the other hand, should the spinal fluid be turbid, contain an undue number of leucocytes, large amounts of globulin and a Gram-positive diplococcus, the child has a pneumococcus meningitis and will die. Even if routine examination of the ears has shown that there is an otitis media, if this is accompanied by signs of meningeal irritation, lumbar puncture is valuable as a diagnostic aid; for either meningism or meningitis may occur together with the otitis. In the case of the former, lumbar puncture is a valuable thera-

peutic aid. When an ear involvement is demonstrated, it is important that the tympanic membrane should be promptly incised. Even if there is no bulging but merely a redness and swelling, the relief of the engorgement by incision is indicated.

Even without misleading signs of cerebral involvement an ordinary pneumonia may be difficult to determine in an infant patient. Careful observation of the type of breathing is probably one of the most helpful means of arriving at a true decision. When the child is quiet, entirely naked, it will be observed that the respirations are shallow and rapid and may run even over 100 a minute. If the child is not observed while it is entirely unclothed, the predominance of abdominal breathing may mislead. Mere inspection of the chest is not sufficient; the child with extensive pneumonia lying quietly and breathing very rapidly and shallowly may appear to be breathing in a fairly normal manner. Very many pneumonia infants breathe in a perfectly characteristic way with an inverted respiratory rhythm. Instead of the normal inspiration, expiration, pause, the rhythm becomes expiration, inspiration, pause. This inversion of the respiratory rhythm is evidence of forced ventilation of the lungs. Oftentimes the forcible expiration is terminated with a definite grunt. This type of breathing is pathognomonic, but unfortunately it does not occur in all the cases of pneumonia. Very extensive consolidations may be present without an apparent inversion of the respiratory rhythm, and even in patients who do show this type of breathing, there may be long periods of the day during which it disappears. Dilatation of the nostrils may be present very often with varying degrees of obstruction to breathing. Usually this dilatation is inspiratory in time, but with the inverted breathing of pneumonia the dilatation is expiratory, and this physical sign is often of great value to the observer when he is in doubt. In marked contrast to what is the case in bronchopneumonia and in severe bronchitis, there is rarely much evidence of respiratory obstruction in lobar pneumonia. The accessory muscles of respiration are not obviously active and the chest is not maintained in the overexpanded position. Subcostal and supraclavicular retraction are rarely seen. The child's facial expression seldom shows the distress and anxiety that so often are seen in bronchopneumonia. The pulse-respiration ratio, so characteristic of pneumonias of older children and adults, does not hold in the pneumonias of infants, and consideration of this ratio is of little aid.

That *pain* occurs with the lobar pneumonia of infants is undoubted. It is especially true of the earlier days of the disease when the pleura is inflamed. In such circumstances cough is followed by a protesting cry and even young infants appear to attempt to restrain the coughing. When the involvement is low down in the lung on the right side, infants old enough to indicate it may complain of abdominal pain. This brings up the same diagnostic question which so often appears in patients of a more mature age: is the primary lesion pulmonary or abdominal? The tendency of pneumonic infants to begin the attack by vomiting, taken together with the pyrexia, leucocytosis and the complaint of pain in the abdomen, may present a most perplexing clinical problem for solution. Often, time is essential to the elucidation of the matter. Only after repeated examination of the chest and the abdomen can a diagnosis be made with certainty. However, it is to be remembered that the abdominal pain accompanying the pneumonia is a referred pain, and that the tenderness elicited by pressure is superficial rather than deep as it is when the abdominal viscera themselves are involved. Furthermore, such rigidity as there is tends to lie above the level of the umbilicus rather than below it, and rigidity is rarely so extreme even in this situation. Some physical signs ought to be found in the chest that will indicate the pulmonary involvement. Cough is a constant symptom, especially in the early days of lobar pneumonia. It is always unproductive and in the first few days it may be painful, but it is rarely harassing and exhausting as it may be in the case of a bronchopneumonia.

The *fever* of lobar pneumonia is not so entirely characteristic in infancy as it is in later life. In general the temperature promptly reaches 103° to 105° F. and remains at that height without much change for a variable period of time (from 5 to 10 days). On the other hand, many infants with lobar pneumonia show very definite oscillation of temperature. There may be a difference of as much as 3° or 4° daily. These oscillations may be regular and the chart may simulate the fever curve of a septic disease, or there may be nothing regular about the swing, and the temperature curve may be entirely without a characteristic contour. In the usual case the temperature drops by crisis, and there may be a fall at this time of as many as 6 or 7 degrees. However, it is by no means true that the fever always terminates in this way. Frequently, in fact usually in the case of infants, the critical def-

ervescence of fever occurs in two stages, sometimes over a period of 36 to 48 hours. A preliminary drop of 2 or 3 degrees takes place; this is followed by a slight rise, and 12 or 24 hours later a second drop takes place, during which a subnormal point is reached. In any case, a subnormal temperature is to be expected for several days following the critical drop. The crisis makes its appearance usually within a week, sometimes as early as the second or third day. Occasionally it is delayed as long as two weeks. When fever persists beyond the ninth or tenth day, a complication should be suspected and sought for, although in many such cases the persistence of the fever results from the involvement of a fresh area of lung and the development of so-called "creeping pneumonia." Very rarely a lobar pneumonia ends by lysis, that is to say that during the course of a somewhat protracted crisis the temperature curve shows some fairly marked oscillation. Once the patient's temperature has subsided, has remained normal for some days and then again rises, it is probable that an empyema or an otitis media has developed. It is a good rule always to examine an infant carefully 4 or 5 days after the crisis of a pneumonia in order to find the earliest signs of a possible complication of this sort.

The examination of the *urine* of a child with pneumonia supplies interesting confirmation when it shows the diminution of excreted chlorides. Usually there will also be some degree of albuminuria. Examination of the urine will also be of value in clearing up any doubt which may exist when the symptoms of an acute pyelocystitis mimic the appearance of a pneumonia, for the presence of large amounts of pus and colon bacilli in quantity will pronounce for a cystitis. However, it must not be forgotten, especially in the case of girl infants, that a pyelocystitis may complicate lobar pneumonia or any other acute febrile disorder during the first two years of life. *Blood* examination reveals a leucocytosis, sometimes of an amazing degree; usually the count is limited to 30,000 or 35,000 white blood cells to the cm. of which a preponderance are polymorphonuclear.

The *physical signs* of pneumonia in an infant are often identical with those found in chests of older patients and they vary according to time of examination. In the stages of engorgement, of consolidation and of resolution, the findings naturally differ. Certain rare cases are reported in which the course and symptoms leave no doubt that the children suffered from pneumonia but in which it was not possible at any time to elicit characteristic physical signs. The

inspection of the child, apart from the characteristic type of breathing already spoken of, often reveals a very definite lag of the chest on the affected side. The difference in expansion of the two sides of the chest is especially noticeable if the child is held erect in a good light with its back to the observer who should stand several feet away from the patient. Auscultation is the most helpful method of physical examination in this disease. In the early stages nothing may be revealed to the ear other than diminution of breath sounds. It is possible to be misled by loud, harsh breathing that may be heard over the lung of the unaffected side. A diminished breathing, especially if it is heard over a lung which has been noticed to be moving poorly, is a sign which should render the observer more than suspicious of a developing pneumonia. Together with this diminution, it is often possible to hear a curious minute clicking râle at the extreme end of inspiration. This has been well likened to the sound that can be brought out by grasping a few hairs just above one's ear and rolling them gently between the thumb and finger. The rapid, shallow breathing of a child who is undisturbed renders it difficult to sense these slight abnormalities in the lung, but very often a fit of crying by developing deeper breathing will make it possible to hear sounds otherwise inaudible. Laying the child on one side hampers the movement of the lower lung and exaggerates breathing in the other. If the observer listens to the uppermost lung and then reverses the position of the child and listens over the other lung, much information otherwise unobtainable can be secured. In the stage of consolidation, tubular breathing is often heard when it is least expected. A thorough examination of the front of the chest may seem to reveal a perfectly normal condition of affairs; occasionally there are a few suspicious sounds to be heard in that lappet of the lung which lies between the heart and diaphragm; when this examination of the front of the chest is followed by an auscultation of the back, most extensive and characteristic evidences of consolidation may be heard. A further peculiarity which may lead to erroneous conclusions arises from the fact that the thin infant chest transmits sounds readily from one side to the other. Listening over the back, tubular breathing or loud râles may be heard at both bases and a double pneumonia suspected when, as a matter of fact, only one side is involved. In this quandary, careful inspection of the chest may be of the greatest aid to a proper diagnosis, and it is

possible that percussion at this stage of pneumonia will be helpful in determining whether one or both sides are pneumonic. But on the whole, percussion is of very little help in determining the presence of pneumonia in an infant chest.

As lobar pneumonia is a specific self-limited disease, no *treatment* other than the institution of measures to support the patient is necessary. Rest, quiet, an appropriate diet and fresh air constitute the essentials. The patient's bed should be placed in an open window screened with muslin to exclude dust and flies. The room should be a sunny one if possible. Covers adequate to keep the child from chilling should be provided, but care should be taken that the common error of overclothing be avoided. The use of the pneumonia jacket is to be deprecated because it does tend to insulate and so overheat the pyrexial patient. A bed cradle is often of service as it keeps the weight and warmth of the bed covers away from the patient's body. Hospital cases should be treated out of doors on a properly sheltered veranda or roof with due regard to the patient's comfort and warmth.

To decide what is the best diet for a pneumonic infant may be difficult, especially in those cases in which diarrhea is a feature. During the first few days of the disease anorexia is the rule, but thirst may be marked; therefore, water can be given freely. Except when there is an acid diarrhea, 5 per cent of lactose may be added to the water or this sugar may be made up with orange juice in the form of high calorie orangeade. Milk tends to produce flatulence and distention; it is therefore well to omit it from the feedings, at least in the earlier days of a pneumonia. Cereal gruels, broths with sago, rice or fine pastes, fruit juices, fruit pulps, toast or toasted crumbs, and an abundance of water provide all that is needed for ingestion during the course of an ordinary pneumonia. Alcohol in the form of brandy or of simple elixir in 5 to 20 drop doses every 4 hours may sometimes prove a serviceable adjunct to other foods.

Apart from these general measures, treatment during an attack of pneumonia resolves itself into the control of symptoms. Of these, the most important is pyrexia which is best combated by hydrotherapy. Antipyrexial drugs are to be avoided. Simple sponging or the use of the tepid or hot bath will often bring an infant's temperature down 2 or 3 degrees. The Preitznitz chest pack (see Methods) finds favor in continental Europe and is certainly effective as an antipyretic measure. When hyperpyrexia with

cardiac weakness and dyspnea appear together, the mustard pack (see Methods) may be applied with gratifying results. As a result of the stimulation attending the high fever in certain cases of lobar pneumonia, sleeplessness and delirium may become extreme. Sodium bromide, 8 grains, in syrup of lactucarium, 1 dram, will sometimes bring about a quiet sleep, but more often recourse must be made to codein $\frac{1}{20}$ grain, by hypodermic, or $\frac{1}{16}$ grain by mouth with sodium bromide, 5 grains, elixir simplex enough to make a teaspoonful. A single daily dose of this sort for 4 or 5 days may be needed. Opium should not be given freely or frequently to control the symptoms in lobar pneumonia. It may be needed to check a harassing cough during the first days of the affection, although the unproductive cough of this stage of lobar pneumonia is often readily controlled by a single daily injection of 1/600 grain atropin for a 20-pound child. The atropin may be given by mouth together with spirits of chloroform and sodium bromide: atropin 1/1000 grain, spirits of chloroform 1 minim, sodium bromide 5 grains, simple elixir and chloroform water equal parts to make a teaspoonful.

Before or during the crisis, signs of cardiac failure may develop and may need treatment. Failure of the heart is indicated by a sudden onset of pallor together with feebleness of the pulse and an increase of the heart's area of dullness to the left. Injections of brandy, 40 minims, camphor 10 per cent in oil 5 to 15 minims, are of great value. In the course of a lobar pneumonia, right heart insufficiency is commoner than heart failure. When the right heart weakens, dyspnea becomes urgent, cyanosis is extreme, and the liver promptly undergoes an increase in size. Under such circumstances, edema of the lung is frequently a terminal complication. Warmed oxygen properly given (see Methods) is a remedy for the cyanosis, and rapid, efficient purgation is indicated. Calomel $1\frac{1}{2}$ grains to a 25-pound child, castor-oil, 3 drams, or $\frac{1}{2}$ ounce of milk of magnesia may unload the portal circulation and reestablish the balance of the circulation. Camphor in oil injections provide a safe and useful remedy. When the right heart is obviously much involved, the mustard pack (see Methods) is useful, but neither its utility nor its efficiency is so great as it is in the cardiac lesions that arise in the course of a bronchopneumonia.

Infants are susceptible to the accumulation of fluid in the pleural cavity. During the first two years of life nearly all pleural effusions are purulent, in the nature of an **empyema**, although on rare

occasions a serous effusion may be encountered. Most often empyemas in infant patients are the sequelae of pneumonias, usually of lobar pneumonias, but sometimes they may follow bronchopneumonias as well. They may also occur in the course of a sepsis or together with an abscess in any locality. The furunculosis of infants is also a potential source of empyema. Any septic disturbance may give rise to an abscess of the lung which in turn by penetration may cause an accumulation of pus in the pleural sac. Most empyemas found in infants are unlocalized; but localization of the purulent fluid does occur either in the fissures between the lobes of the lungs, or at the base between the diaphragm and the lower surface of the lung, or at the apex, or along the hollow of the chest in the paravertebral curved space. The detection of such localized empyemas is a matter of great difficulty, although it is rendered somewhat easier by a study of radiograms of the chest and of the fluoroscopic appearances of the thoracic contents.

By far the greatest number of empyemas follows lobar pneumonia. Therefore a knowledge of a precedent pneumonia, or of an antecedent illness which can reasonably be supposed to have been a pneumonia, is of great service to the attending physician. Acute tonsillitis, affections of the nasopharynx, osteomyelitis, otitis media, scarlet fever, diphtheria, and measles, any of them may be precursors of an empyema in an infant who has given no evidence of a true pneumonia. The organism which is most often etiologic in this condition is the pneumococcus, but staphylococci and streptococci and bacilli are all to be incriminated at some time or another.

The revealing evidences of an empyema are a history of a pneumonia or the presence of a septic focus; a cough which may be painful in the beginning, dyspnea; pyrexia with a septic swing to the temperature curve; and in neglected cases with a history of long illness, sweating, anemia, venous engorgement of the vessels of the neck and face, clubbing of the fingers, albuminuria, and cylindruria. In such cases the temperature curve is essentially that of a sepsis. The most striking physical sign which confirms the inferences to be drawn from these symptoms is on inspection a diminished movement of the chest on one side. (This is especially evident in the case of young infants.) It may not be seen while the child is breathing quietly, but the exaggerated respiration brought on by crying develops the sign clearly. Inspection also shows an altered position of the heart's impulse which is drawn out of its normal site by the traction of

the lung of the unaffected side. The earliest sign of a left-sided pleurisy in an infant is the shifting of the maximum cardiac pulsation to a point below the ensiform cartilage. In infants who have fluid in the right chest, the heart is shifted upward rather than to the left and the maximum impulse may lie as high as the fourth or even the third space. Late in the course of an untreated empyema, bulging of the ribs on the affected side may be seen.

Palpation reveals little that aids in the diagnosis of the condition in an infant patient, although if the cry fremitus is not to be detected over a large part of one side of the chest, this fact furnishes confirmation of other signs of a pleural effusion.

Percussion shows an impaired resonance which reaches high up into the axilla. This dullness slopes downward to meet the cardiac dullness in the front, and behind it runs to the edge of the spine. In the presence of a pleural effusion, the diaphragm on the same side becomes embarrassed and loses tone. Under these circumstances a distended stomach may push upward until the resonance is delimitable in the axillary line as high as the fourth or even the third rib. Therefore, instead of being negative evidence of the presence of a left-sided pleural effusion, a high-lying stomach resonance is suggestive of this very pathological condition, for normally the tension of the diaphragm should prevent this upward shift of gastric resonance. While this resonance may reach as high as the fourth or third rib laterally, it rarely goes upward beyond the level of the eighth rib behind. Above this high-lying resonance when pleural effusion is present, there will be an area of percussion dullness due to accumulated fluid, and still higher on the chest toward the apex of the lung the hyperresonance of Skoda, which is due to a compression of the lung tissue, will be elicited. When the effusion is on the right side, the picture is altered by the fact that instead of the hollow stomach, the lung is in contact with the solid liver. Due to a hypotonus in the right diaphragm, the liver dullness rises higher than normal and blends on this side with the flatness produced by pus lying in the right pleural cavity.

Over the lung, apart from the effusion, breath sounds are diminished, but instead of the normal note, the percussion reveals liver dullness blending at a point higher than normal with the flatness of accumulated pus. Percussion over the lung tissue on the affected side shows an impaired resonance. Auscultation of the compressed lung reveals much that aids the diagnosis of empyema. Over the lung on the side of the effusion breath sounds are dimin-

ished and expiration is exaggerated, but true bronchial breathing is rarely heard except at the apex, along the spine, and immediately above the flatness caused by the fluid in the chest.

In the later stages of an empyema when pulmonary elasticity has been lost and the accumulated fluid exerts direct pressure on the heart, diaphragm, mediastinal contents and lungs, the diaphragm will be pushed down. The stomach resonance and liver dullness will then lie lower than normal and the ribs and intercostal spaces on the affected side will be bulged instead of being retracted, a clinical picture quite in contrast with what is to be discovered on examination of the chest during the earlier stages of the affection.

The *treatment* usually advised for empyemas is surgical incision with rib resection and drainage. This is a very unsatisfactory method of treatment for young infants whose lungs are too weak to reexpand after collapse. For these the writers have found repeated aspirations to be an effective procedure. An ordinary 30 c.c. or 50 c.c. Luer syringe with a needle of short bevel is the only apparatus needed. Fluoroscopic observations of the chest enable an accurate check to be kept on the progress of the case. (See Methods.)

The development of an extreme **pneumothorax** is almost unheard of during the early years of life although it has been known to follow after a foreign body has ulcerated its way from a bronchus into the pleural cavity and so established a communication between the bronchus and the chest wall. Usually the condition is of **pyopneumothorax**, for in all the cases some pus is present with the air. Evidence of localized collections of air in the pleural cavity may appear after exploratory puncture if the aspirating needle has passed into the lung and entered a bronchus. The signs indicative of a massive pneumothorax are displacement of the heart, bulging of the intercostal spaces, immobility of the lung on the diseased side, and the appearance of an area of tympany where normal lung resonance should be. The coin test produces a clear bell-like sound heard in the stethoscope placed anywhere over the pneumothorax. An infected mediastinal gland may adhere to a bronchus and later suppurate. Under such circumstances the glandular abscess has been known to perforate the bronchus and to set up a pneumothorax.

The *treatment* of the massive form of pneumothorax is evacuation of the accompanying pus and treatment of the primary pulmonary disease by fixation of the chest and the use of opiates to assure rest. Localized accumulation of air absorbs spontaneously.

When the air-cells of an infant fail to expand and to develop respiratory function at birth, the child is suffering from **congenital atelectasis.** Ordinarily the stimulation of the cold external air on the skin establishes inspiration and the respiratory centers are automatically put to work. Crying which is in effect but a form of forced respiration further stimulates it. Atelectasis is not to be feared if an infant is crying lustily. Prematurity and congenital weakness are the usual causes of a lack of expansion of the lungs. Prolonged labor which has been exhausting to the infant, or undue pressure on the child's head is contributory; either may exhaust the child's nervous system or produce a condition of congestion in the cerebral veins that interferes with the proper functioning of the brain. The embarrassment that arises in the pulmonary circulation when a congenital defect of the heart is present is another possible source of atelectasis. The persistent patency of the ductus arteriosus or of the foramen ovale, by interfering with the circulation in the lung, can contribute to maintain the fetal condition of the air-cells.

The diagnosis of the condition is to be made more upon the appearance of symptoms than upon physical signs, for the latter are few and uncertain. The child is almost invariably small and ill-nourished and obviously weak. It shows great pallor, sometimes with a little cyanosis. Very often the pallor is broken from time to time with an attack of urgent cyanosis. The child is cold to the touch and the temperature registration is subnormal. The swallowing is difficult; very often an attempt to swallow brings about a cessation of breathing that endures for two or three minutes. This apnea is attended by a further wave of cyanosis. Inspection shows the chest to be moving very superficially, so much so that on some respiratory efforts it sinks in rather than expands; and for considerable periods movements of the chest may be imperceptible. When the atelectasis is extensive, as may happen when there is a congenital obliteration of the bronchus, the percussion note may be dull; most often there is enough air in the lung to give moderate resonance. The breath sounds are rarely bronchial in type; they may be absent over large patches of dullness, but they are chiefly feeble and vesicular in character, and auscultation of the parts reveals the weak sounds of an ineffective inspiration. It is important to decide promptly if a child showing such a clinical picture is suffering merely from a deficient expansion of the lung. Careful scrutiny of the possibilities of cerebral hemorrhage should

be undertaken (see page 122) and if there is any chance that an intracranial hemorrhage has taken place, immediate steps to meet this condition should be taken. Lumbar puncture should be done and, if need be, operation resorted to. The fact that great distention of the abdomen may contribute to the respiratory embarrassment of the newborn must not be overlooked. Attempts to stimulate respiration in the presence of either cerebral or abdominal causes of apnea can lead only to an exaggeration of the condition.

After all discharges and accumulation of mucus have been carefully cleared from the nostrils, mouth and throat, the next and most important measure of treatment in dealing with a congenitally atelectatic infant is through methods of external stimulation, mechanical and thermal. The alternate bath of hot water 110° F. and cold water 80° F. is usually the first measure employed. The water is poured into two tubs placed side by side and the child is passed from one to the other. In the meantime, vigorous friction or mild flagellation with a wet towel is applied to the body. The proceeding is continued until the child begins to breathe well or until it is obvious that no further result can be obtained. At this point, if the child is still apneic, artificial respiration should be undertaken, preferably by the Sylvester method. (See Methods.) The Schultze method may be used; in skilful hands it is very effective, but unless applied with the greatest care, it is easy to bring about a fatal injury. When the breathing has become established, the usual regimen appropriate to the care of the premature or weak should be instituted. The child should be placed in a premature jacket in a warm cotton nest in a basket and should be surrounded by hot bottles or warmed with an electric heating pad. The air should be warm and moist but pure, and the room should be well ventilated. Too often the premature and weakly child is placed in the bottom of a basket in a position such that fresh air cannot reach its nostrils, and the room is often so insufferably hot that the temperature works to the detriment rather than the benefit of the child. A room temperature of more than 65° should not be allowed; 62° is better.

The tendency of the child to stop breathing when it attempts to swallow must be remembered and if this symptom is annoying, the feedings for some days must be by gavage. Intramuscular injection of 3 to 5 or 6 minims of brandy should be given every four or six hours in order to stimulate the child. Change of

position is of advantage, but these changes must be made with the greatest care; overhandling of the infant is exhausting to it and is to be deprecated. Once respiratory vigor is established, those measures indicated for the care of weakly or premature infants are more likely to rescue the child than are vigorous efforts to develop full respiration.

Acquired atelectasis is the result of collapse of the lung. Most often it follows bronchial, tracheal or laryngeal obstruction due to aspiration of a foreign body or to the blocking of a bronchus by an accumulation of viscid secretions. It is essentially a disorder of early life and, apart from the results of aspiration of a foreign body, is rarely found in any but weakly children. As a complication of pleural effusion (empyema) an analogous collapse is the rule. The symptoms brought about by the condition are increased respiratory rate, dyspnea and cyanosis. These occur in the course of a bronchitis or laryngitis and may readily escape observation. The physical signs are more revealing than the symptoms. Diminished breathing or bronchial breathing and increased cry fremitus will be found, although if the air cell collapse is great, it may be possible to discover that no air is entering the alveoli. Except in very weakly infants, this condition is of little clinical importance, for the area of collapse is rarely big enough to bring about a fatal interference with respiration, and with the improvement of the causative conditions the collapsed area re-expands and the child goes on to recovery.

The *treatment* of the condition when diagnosed includes all those measures that may render respiration more free. Change of position, removal of restraining bed-clothes, injections of brandy and adrenalin, the inhalations of oxygen, and mechanical stimulation by friction or flagellation of the chest, any or all of them may be indicated in an individual case. If opium or any other respiratory depressant is being used, it should be discontinued at once. Atropin may be given by hypodermic injection in a single maximum dose appropriate to the size of the child: $\frac{1}{300}$ grain may be given to a 20 to 25 pound child with advantage.

CHAPTER XIII

DISEASES OF THE DIGESTIVE TRACT

Those diseases of the alimentary tract in which *diarrhea, vomiting* or *nutritional disorders* are predominant features have been dealt with in Part I under appropriate headings.

Malformations of the esophagus are met during infancy. In the upper half of the gullet certain abnormalities incompatible with life are encountered at birth. These are uniformly rapidly fatal. In one variety the pharynx has a blind end terminating about the level of the cricoid cartilage. Sometimes there is a lateral fault in this sac which leads into the trachea; in other cases parts of the esophagus have failed to develop beyond the solid cord epithelial stage and a simple atresia is the result. This form of obstruction is usually found in the mediastinum where it is inaccessible to surgical procedure.

The esophagus may be occluded by the pressure of abnormal structures from without. An aberrant subclavian artery has been known to act in this way.

Older infants may acquire an obstruction of the esophagus following the ingestion of strong alkalies. For the most part, however, such accidents are fatal; when they are not followed by death, the trauma to the gullet usually results in partial scarring only and is amenable to treatment. It may be necessary to perform a gastrotomy and to avoid use of the esophagus until the process has become thoroughly healed. Once the scarring has become firm the passage of graduated bougies will achieve sufficient dilatation to permit ingestion of food. Food is best given in a semifluid state; solids and fluids are both badly taken. The greatest care is necessary in the use of the bougies, for penetration of the esophagus may take place.

Foreign bodies such as coins, large beads and masses of food sometimes lodge in the esophagus and cause an obstruction. To remove these, especially when they are hard in nature or of irregular form, demands great skill; wherever possible the attempt should be made only after the foreign body has been viewed through an esophagoscope.

Diverticula of the esophagus may be of congenital origin or they may be acquired through the lodgment of a small foreign body and the subsequent stretching of the tissues. The acquired form is negligible as the source of diverticula during infancy. The congenital type of pouching is often accompanied by some narrowing below it, and the symptoms are usually those of a stenosis. However, food will accumulate in the wide part of the tube and the amount of food regurgitated will be larger than is the case when the narrowing of the esophagus is uncomplicated by a diverticulum.

Defects and deformities of the stomach are rarely met with. In the files of the University of California Hospital a description is recorded a deformity of the pyloric mucous membrane of such nature that it acted as a valve obstructing the pyloric end of the stomach; it produced symptoms which mimicked those of a pyloric stenosis.

Congenital pyloric stenosis is a condition well recognized and much written about. Its classical symptom is persistent vomiting which rarely begins to be apparent before the end of the third week of life; it is obstructive in type, cumulative and forceful. Sometimes the earliest vomitus may contain a quantity of blood and suggest the presence of an ulceration. Together with the vomiting there is constipation, diminution of urinary output and anhydremia with rapid loss of weight, emaciation and the early supervention of mild acidosis. The stomach is obviously dilated, the upper portion of the abdomen balloons out in striking contrast to the narrowed lower part, and a characteristic wave is to be seen traveling over the dilated stomach. This wave rises at the left costal margin where it appears as a localized bulging which passes slowly downward and to the right to disappear on the right side. This motile bulging reproduces itself about 20 times a minute. It is best observed immediately after the child has ingested a meal. Often it is possible to palpate a tumor—the mass of the hypertrophied pyloric sphincter.

That pyloric stenosis is truly of congenital origin there can be no doubt, for the study of stomachs of certain stillborn babies proves that it can be present in antenatal life. The condition is undoubtedly one of overgrowth of the muscular and fibrous tissue about the pylorus and pyloric antrum. The obstruction is, to some degree at least, due to a functional spasm of these hypertrophied tissues, for the obstruction in any given case is not always of a like degree at all times. There may be a period at which certain meals may

pass freely, followed by another during which no food gets beyond the pylorus. The success that has followed the use of thick feedings also suggests that the obstruction is more often the result of spasm than it is of mechanical blocking. There is a group of cases constituting the so-called **pylorospasm** in which the symptoms of obstruction are identical. These show all the physical signs of pyloric stenosis except the presence of a demonstrable tumor. In these cases there is no hypertrophy of the pyloric structures and the clinical symptoms respond promptly to appropriate dietetic treatments of which the most certainly effective is the institution of thick farina feedings after the method of Sauer.

The *treatment* of a case of pyloric stenosis will vary with the condition of the patient and the length of time which has elapsed since the onset of the symptoms. With a child who has only recently developed evidences of the disorder, one who is in good condition, strong and not losing weight rapidly, it is permissible to temporize. A trial should be made of the thick feeding. (See Formulas.) Small amounts at 4 hour intervals should be given in the beginning and if this form of food is well tolerated and if the vomiting ceases or ameliorates to a considerable degree, larger amounts may be used. If the child is visibly dried out, intraperitoneal or subcutaneous injections of appropriate amounts of fluid (Ringer's solution or normal saline solution) should be made at the outset of the treatment. It is not to be expected that an anhydremic child will digest and properly utilize food. If the vomiting does not cease promptly or if the general condition of the patient remains poor, prompt recourse to surgical intervention should be made.

It is hardly fair to expect the surgeon to achieve results when the patient has become weakened and depleted by prolonged starvation. In the Fredet operation, sometimes called the Ramstedt, we have a perfectly simple and effective surgical means for overcoming the condition. In this operation after abdominal incision, the pylorus is delivered through the wound. A simple longitudinal cut is made which passes through the serous and muscular coats of the hypertrophied mass but does not injure the mucous layer of the pylorus. If the pylorus is grasped by an assistant who turns it over and holds it, a comparatively bloodless area can be found where the incision can be made without the need for tying any vessels. In performing the operation, experience is

necessary in order not to nick the thin-walled duodenum just below the tumor. However, if this part of the intestine is incised, no great harm is done, for it is an easy matter to put in one or two stitches and thus close the wound. A skilled surgeon operating on patients who are not already moribund will be successful in a great majority of cases if he uses the method of Fredet, popularized by Ramstedt.

Following operation, the child should be placed in a semierect position in bed. Four hours after the operation and every 2 to 4 hours thereafter for 12 hours, an ounce of 5 per cent lactose or maltose solution may be given by mouth. At the end of 12 hours, breast-fed babies will usually nurse, but for another 24 hours the time of their nursing should be restricted to 5 or 6 minutes. After that they may be treated as healthy infants. Babies receiving formulas may have a feeding of dilute, sweetened condensed milk, (1-20) 12 hours after operation; this is to be repeated every 4 hours for another 12 hours, when a stronger formula (1-16) may be tried and continued at 4 hour intervals for another day. After this time, normal formulas suitable for the age and weight of the child may be used. The thick farina feeding is well tolerated and is of use. It is best, however, to begin the feeding of milk formulas by using a dilution somewhat greater than would be appropriate for a normal baby and to increase the concentration from feeding to feeding until the full strength mixture is given.

Acute dilatation of the stomach may occur in a baby after operation for pyloric stenosis or after any other abdominal operation. Occasionally it happens in the course of a pneumonia or of an acute enteritis. Under these circumstances there will usually be an accompanying dilatation of the intestines. A fatal issue may supervene rapidly because of the crowding of the diaphragm and the embarrassment to the respiration and the circulation. On the appearance of symptoms of acute dilatation, immediate injection of ⅓ c.c. of surgical pituitrin may be effective; following the injection washing out of the stomach and of the bowel with 5 per cent sodium bicarbonate solution is indicated.

Atresias and **stenoses of the small intestine** occur and may be responsible for symptoms of bowel obstruction shortly after birth. When the narrowing is high up in the duodenum above the point of insertion of the pancreatic and liver ducts, the symptoms are identical with those seen in pyloric stenosis but they supervene immediately after birth; palpation reveals no pyloric

tumor. Among the patients of one of the writers there is a child now 4 years of age who was born with an atresia of the duodenum and upon whom a gastroenterostomy was performed on the fourth day of life. The infant made an uninterrupted recovery and has remained perfectly normal since, growing and developing without untoward incident.

Atresias of lower parts of the small intestine are usually multiple and therefore surgery is powerless to relieve them. However, it is impossible by physical examination to decide that they are not single, and a newborn infant with evidences of anatomical obstruction should be given the benefit of an exploratory incision with the hope that an operation may be of help.

A very unusual form of bowel obstruction of anatomical origin is sometimes seen when an **intraileac cyst** springs from the mucous membrane of the ileum within a few inches of the ileocecal valve. Such a cyst may change position and produce intermittent obstruction, acting at times as a ball valve and occluding the outlet of the ileum.

Narrowings, atresias and stenoses of a part or all of the large intestine may take place with the production of symptoms of bowel obstruction. The same sorts of pathologic events may occur in the anal canal where they are much more common than higher up. The most usual atresia about the rectum is the result of persistence of the normal membrane which forms between the inverting proctodeum and the descending hind gut. Such an obstruction should be discovered in the usual inspection of the newborn infant. About two centimeters from the anal orifice the finger will encounter, on palpation, a thin, flexible membrane, which is readily opened by simple incision.

Other obstructions in this region are of the nature of true atresia or of congenital malformation of the primitive cloaca. In the cases of atresia brought about in this way, the whole of the anal canal may be a solid cord, which on palpation will be hard and resistant without any apparent hollow viscus above it. When such a state of affairs is discovered, there is no alternative other than to summon a surgeon in order that a colostomy be made and the intestinal contents emptied through the incision at the iliac fossa. If this atresia of the rectum be the only deformity of the intestine, after some months or a year an attempt may be made to construct a rectal canal leading out at the anus. In the congenital malformations which involve the cloaca, the rectum may open into

the bladder or the urethra of boys or into the bladder or vagina of girls. Very little can be done when the urinary tract is involved. When the opening is into the vagina, the repair of the defect may well wait until the child is 7 or 8 years of age before an attempt at reconstruction is made.

Congenital giant colon (megacolon) is not a very rare disease. Many cases have been described since 1886 when Hirschsprung fixed the attention of medical men on the condition by the publication of a monograph on the subject. Much controversy has been aroused as to the nature of the malady. Hirschsprung's view was that the pathologic conditions were the results of an antenatal structural anomaly. This view is supported by the appearance of the condition in stillborn infants. One of the writers has recorded a case of Hirschsprung's disease discovered in a newborn child. The gigantism of the colon was perfectly developed and the pathologic findings differed in no way from the conditions found in older children with the same affection.

Usually, obstinate constipation is a symptom of megacolon; this may occur periodically in alternation with foul diarrhea. Occasionally obstruction of the bowel has taken place from the lodgment of a fecal mass in a narrow point of the intestine. This is most likely to happen in those cases in which the dilated sigmoid ends abruptly at a narrow rectum. All the patients with megacolon suffer from attacks that are obviously of a toxic nature. Lassitude, pallor, sweating and weakness are prominent symptoms, but these pass off during the diarrheal periods. The physical sign on inspection is the outline of a largely dilated colon, and often there is a visible peristalsis which appears in the line of the transverse colon; the movement is from right to left; or the wave may be visible over the loop of the enlarged redundant sigmoid when it will appear double; one undulation, the upper, will then move from left to right, while the lower sweeps from right to left. Palpation of the abdomen will often reveal large hard masses of fecal accumulation in the dilated gut, and a radiogram taken after a bismuth enema has been given reveals the dilatation and redundancy of the colon in detail.

Medical *treatment* is of little use. Purgatives and high enemas may accomplish efficient evacuation. A low enema of mineral oil is sometimes a help, and when distention and retention of colonic contents are great, an injection of pituitrin may produce partial evacuation. The only effective treatment is surgical. Excision of

the giant colon should be done as soon as the diagnosis is certain, provided that the child's physical state permits. Delay is followed by the development of increasing toxemia and nutritive disturbances which may render surgery useless.

A great many babies suffer from **anal fissure.** It is one of the commonest causes of intractable crying. The infrequency with which the anus is subject to routine examination is the reason for the generally held opinion that fissure is uncommon during babyhood. Careful examination of this region in every infant will disclose evidence that quite the contrary is true. It is necessary to obtain a good view well up into the anal canal, for a high lying fissure may easily be missed, especially when there is much hypertrophy and spasm of the sphincter. Much pain and distress that is attributed to colic is in truth the result of fissures and of tears about the anus; and many a crying, sleepless infant can be made tranquil after the discovery and treatment of a painful fissure. Fortunately the tendency is to spontaneous recovery.

The original trauma most often comes from the overstretching of the anal canal by a large hard fecal mass. Such masses are more apt to occur in the intestines of bottle-fed children; therefore fissures are more frequently found in such infants. Damage from the hard point of an enema tube also contributes to produce these tears, especially in the case of the newborn. For this reason the soft all-rubber "ear and ulcer syringe" is preferred to the hard pointed rubber bulb apparatus usually sold as an infant's rectal syringe.

The damage to the mucous membrane is soon followed by a *spasm of the anal sphincter* and very often, if this spasm continues long enough, hypertrophy of the muscle is produced. The hypertrophy and painful spasm of the anal sphincter may continue long after the fissure itself has been spontaneously healed.

Children with fissure in ano pass stools with difficulty and often after a great deal of pain. On the other hand, defecation may be painless and the attacks of recurrent pain may seem to come entirely from the abdomen. Apparently the ingestion of a meal starts peristalsis which, rolling along the gut, reaches the anus and produces sudden sharp painful contraction of the sphincter. The child, in distress, draws up its legs and screams. Spasms of pain and attacks of crying may succeed one another at short intervals for half an hour or more after each meal. These manifesta-

tions may easily be taken for intestinal colic of the ordinary type, but no amelioration follows those measures which are usually successful in checking colic. The seizures will promptly disappear after efficient treatment of the anal condition.

This *treatment* of fissure in ano consists of gentle digital dilatation of the sphincter ani followed by free application of a soothing ointment. It is sometimes well, before applying the ointment, to make a single application of 20 per cent nitrate of silver solution to the ulcerated surface of the fissure. Any soothing ointment may be used. One that has proved efficacious is made up as follows: ichthyol 10 per cent, extract of belladonna ½ per cent, simple cerate 25 per cent, and enough cold cream or petrolatum to make 100 per cent. The mother or nurse is supplied with a finger cot and is instructed to anoint the finger thoroughly with the ointment, pass it well into the rectum, and to make a digital dilatation at each application; this should be repeated morning and evening for 4 or 5 days by which time the symptoms of the condition should have disappeared.

Proctitis is a frequent complication of acid, excoriating diarrheas. It is usually present in some degree whenever an excoriation of the buttocks is to be found. The slight proctitis of this condition is amenable to small alkaline injections; an ounce or two of warm 5 per cent bicarbonate of soda solution may be thrown into the rectum once or twice a day for two or three days. When the rectal irritation is unaccompanied by acid stools and when there is much mucus in the evacuations, similar sized injections of ½ of 1 per cent of aluminum acetate solution have proved very effective. When proctitis is accompanied by much tenesmus, after a preliminary washing with an alkali, a few ounces of warm, well-boiled starch solution may be put into the rectum with advantage. Efforts should be made to have it retained as long as possible. Some authorities recommend the use of opium together with the starch solution, but the latter alone is quite as effective in relieving the distress.

Small **polypi** spring from the mucous membrane of the lower sigmoid and rectum and frequently cause the appearance of bright red blood in the stool. These hemorrhages may be small or profuse and they are often accompanied by "bearing down" and straining while at stool and between evacuations. Babies who show this peculiarity and the presence of bright red unchanged blood in their movements or on their napkins should undergo digital and proctoscopic examination. Oftentimes polypi will be found accountable;

these are readily removed by surgical means and they should be promptly dealt with when discovered.

Acute acquired intestinal obstruction from whatever cause, is characterized by a sudden onset with pain and shock, persistent intractable vomiting going rapidly on to fecal emesis, and retention of flatus with abdominal distress. Often intestinal peristalsis is visible. The facies denote shock, the pulse is rapid and thready, the pupils are dilated, the skin is pale and the eyes are sunken. The expression is one of extreme weariness. Toxic lethargy emphasizes the gravity of the patient's condition.

Internal **hemorrhoids** are not common at the pile-bearing area of infants. Occasionally, however, a small vessel at the mucocutaneous margin may develop a thrombus. This condition is always accompanied by a sudden accession of severe pain and tenderness which lasts but a short time. Surgical intervention is rarely necessary, but should the condition persist, it is a simple matter to shell out the clot through a small incision and so relieve the child.

Intussusception which is the commonest form of acquired obstruction during infancy is marked by certain revealing symptoms not usual in other forms of bowel obstruction; these result from the peculiarity of the condition. The invagination of one portion of the intestine into the other begins the morbid process and, as the invagination proceeds, the mucous membrane of the gut is damaged; as a result of this damage some bleeding occurs, enough to mix thoroughly with the mucous secretions of the bowel and to give rise to several evacuations of bloody mucus, the appearance of which has been well likened to currant jelly. This particular form of bloody stool together with abdominal shock, pallor, pain, and vomiting is pathognomonic of intussusception.

The infolding of the bowel, with the local swelling that follows it, produces a typical tumor mass. In the earlier days of such an obstruction the belly wall is not distended; on the contrary it is usually flaccid and the tumor is readily palpable. If the patient be seen within the first few hours after the onset of vomiting, the mass will be located lying in the upper part of the ascending colon or about the hepatic flexure. In this situation the mass may be difficult to feel as it may lie hidden behind the liver. Later the intussusception can be found at the splenic flexure or somewhere in the descending colon. Only after many hours will the mass come down low enough to be palpable in the rectum.

The movement of the gut downwards carries with it the caput coli, and as a result palpation over the right iliac fossa leaves a sense of unnatural emptiness. Rectal examination under a light anesthetic also reveals an emptiness here and the absence of the appendix which normally should be readily felt by bimanual palpation. If the invagination has proceeded far enough, the tumor itself may be felt by bimanual palpation with one finger in the rectum, provided the mass lies below the level of the umbilicus.

Once it is certain that the obstruction is due to an intussusception, an attempt at reduction should be made by the hydrostatic reposition method of Hirschsprung. This maneuver is best accomplished under the operator's eye after abdominal incision. This method is especially of value in those cases that come under observation late. Resection of the intestine should never be resorted to until a thorough trial has been given the Hirschsprung method.

An important but comparatively unusual condition which must come into consideration when we are dealing with abdominal diseases of children is *chronic intussusception.* The onset of this disorder is sudden, approximating the classical onset of the acute intussusception; but the symptoms are decidedly different. In this chronic form, instead of the picture of complete obstruction, there is a subsidence of symptoms during the course of two or three days in which the vomiting, pain, and shock become progressively less; instead of the characteristic blood-stained currant jelly-like stools of the acute form of the disease, bowel evacuations are passed which are bloodless, or, at most, only slightly blood streaked. This condition of affairs remains, perhaps with slight temporary exacerbations from time to time, until several weeks have passed. After this period, progressive wasting sets in and becomes the most striking feature of the malady.

Abdominal examination reveals to the palpating hand the characteristic sausage shape tumor, less tender than that found in acute intussusception; this tumor is usually larger at one extremity than at the other, changes consistency under the palpating hand which finds it lying in the line of the colon on the right side under the liver. By manual rectal examination at this time, an abnormal feeling of emptiness in the right iliac fossa will be discovered. The findings together with a lack of fever, and the evidence of progressive wasting and recurrent periods of temporary bowel obstruction constitute the characteristic picture.

Tuberculous peritonitis is the affection most often confused.

The slow onset of the tuberculous condition, and the malaise of the tuberculous child are such that they should afford an adequate differentiation from chronic intussusception; the latter always begins acutely, and the characteristic tumor is felt quite different in shape from the hard caseous combinations of gland, swollen peritoneum and omentum to be found in cases of tuberculous peritonitis.

Chronic intussusceptions occur as early as the first year and are to be found at any time throughout the duration of childhood. Several weeks may elapse between the onset and the termination of the case either by operation after diagnosis or by death with progressive malaise if the condition be overlooked.

It is rare to find any great swelling, or much adhesion, or tendency to slough in the tumor of a chronic intussusception; therefore resection seldom becomes necessary, and the prognosis is good because the intestinal infolding yields readily to methods of simple reduction.

Shortly after birth, obstruction of the bowel may occur from the lodgment of *inspissated meconium*. The condition may be difficult to diagnose from an obstruction due to congenital stenosis or an atresia of the small bowel. However, the symptoms are rarely so urgent and give way after a few hours if thorough flushing of the bowel with 5 per cent or 10 per cent sodium bicarbonate solution is undertaken.

Foreign bodies may lodge in the intestine of older infants and cause obstruction, or a pin may be swallowed which later may perforate, produce a peritonitis and lead to a paralytic ileus. The history of the case and the aid of radiograms will make clear the diagnosis after such accidents.

Upon the diagnosis of other forms of intestinal obstruction, operative procedure alone will be of avail. The important thing is to make an early diagnosis and as soon as the decision is taken to operate without hesitation, because if relief of the obstruction be delayed, the condition is almost certainly fatal.

Acute suppurative **appendicitis** is unusual in the first two years of life although it does occur as early as the end of the first month. When the condition is present in the abdomen of an infant, it is diagnosable only in the rarest instances because abdominal pain, vomiting and low degrees of fever are common manifestations during infancy.

One infant known to the writers developed a peritonitis follow-

ing a rupture of a suppurative appendix in its twentieth month. A review of the history of this case revealed the fact that from the time the child was 8 or 10 months of age it had suffered from seizures of abdominal colic sufficient to wake it from sleep. As it grew old enough to stand in its crib, it would lean over during these attacks of recurrent pain and press the edge of the railing across the lower abdomen, a procedure which seemed to give it relief. These attacks were very short, not more than 5 or 6 minutes in duration, but they would recur several times during the night for perhaps a week and then disappear. They were attributed to indigestion and the temperature was not taken nor was a blood count made until after the final explosion of the appendix. Another symptom was elicited from the history in this case which, while suggestive, may be misleading: this was pain with urination which came on at the time of the attacks of abdominal colic. It must not be forgotten that certain boy babies who have a narrow urinary meatus may acquire a painful distention of the bladder because of difficulty in emptying that viscus, and that symptoms analogous to those suffered by this child with appendicitis may occur as a result of distention of the bladder and vesical spasm.

Another infant who developed a peritonitis after a ruptured appendix, had some two months before, a severe seizure of what was apparently abdominal colic. This child was more than 2 years of age and did not vomit and was able to indicate the site of the pain, which he referred to the right side of the belly above the umbilicus. At the time of this attack of colic, careful palpation of the abdomen revealed no rigidity and no tenderness. However, when the abdomen was opened after the appendix had exploded, it was discovered that the boy's cecum lay very high and that the appendix contained a concretion larger than a kernel of a peanut.

A third infant, twenty-two months old, who developed a peritonitis as a result of a ruptured appendix, had suffered several attacks of abdominal pain with some tenderness, the first appearing at the eighth month, but all the evidence was that the morbid process lay somewhere about the sigmoid. On operation, a very long appendix was discovered which stretched across the pelvic brim and was adherent at its tip well over on the left side. Rupture had taken place at the point of adhesion with the formation of a localized abscess.

It is improbable that a physician will see a case of appendicitis in an infant under two years of age before the appendix has rup-

tured and the picture becomes one of generalized peritonitis. Localization after rupture in an infant abdomen is hardly to be expected. In the instance recorded, it was a happy incident. However, as a matter of precaution, recurrent seizures of abdominal pain during infancy should not be considered to be dietetic until after repeated physical examinations of the abdomen have been made and the leucocytes enumerated. Vomiting, abdominal distention, constipation and shock with localized abdominal rigidity together with a leucocytosis may permit diagnosis before peritonitis has developed. By way of *treatment* a prompt drainage of the peritonitis which follows ruptured appendiceal abscess is usually rewarded by the patient's recovery.

Diastasis of the abdominal recti is a defect commonly met during babyhood. It contributes in producing the prominent abdomen of many infants and young children. The condition also plays a rôle in the production of deformities of the chest cage, Harrison's groove especially. The nonunion of the rectus muscles deprives the costal margin of its normal external central anchorage; as a result the diaphragm acts without proper muscular antagonism and pulls in the soft ribs along the line of the anterior part of its insertion—a state of affairs that is exaggerated by any factor which tends to prevent free filling of the lungs. Later, when infants have assumed the erect posture, diastasis of these muscles is a potent factor in the development of visceroptosis and the spinal deformities, especially swayback, which result from dropped viscera.

Certain forms of bowel obstruction happen in the newborn as the result of developmental abnormalities about the umbilicus. For instance, there may be a **hernia into a persistent umbilical sac** with fixation and obliteration of a part of the intestine; or a prolapsed loop of intestine may be pinched at its base and incarcerated in such a sac.

Hernias into the thorax may occur through abnormal openings in the diaphragm.

Mesenteric or omental faults may include and strangulate loops of intestine and thus produce symptoms of obstruction shortly after birth, or the appearance of herniations of this sort may be delayed until weeks or months later when the obstructions may become apparent. They are then difficult to differentiate from other **acquired obstructions.** During infancy the most important of these acquired obstructions arises from a persistence of Meckel's diverticulum, an intussusception, a volvulus, a peritonitis, or from

the presence of abnormal tissue bands which may constrict the intestine or present abnormally placed passages through which the gut can herniate.

Congenital hernia of the cord is a rare condition and one due to imperfect closure of the abdominal walls with protrusion of the abdominal viscera. It is often associated with harelip and other congenital anomalies and it is analogous to ectopia of the bladder. If small, it may be treated by simple strapping; otherwise operative procedures are necessary to secure coaptation of the abdominal walls.

The ordinary **umbilical hernia of infancy** is equally frequent in the two sexes. It is due to the thinness of the tissues, or to an abnormally large umbilical opening, or to both. Since the vein lies at the upper part of the cord and is less firm than the artery, the herniation usually occurs at this part of the ring. Spontaneous cure is the rule and strangulation of an umbilical hernia is extremely rare because, except in quite large ruptures, the sac contains omentum only. The condition is frequent in badly nourished, weak infants and in the premature. The ready protrusion follows from the laxity and poor turgor of the muscular walls, the loss of subcutaneous fat, and the various respiratory and gastrointestinal conditions which tend to increase intraabdominal pressure. The prime means of attaining cure include all measures directed to improving nutrition. Strapping by means of adhesive tape, if the whole umbilical region is infolded, will usually result in a cure within a few months if undertaken early (see Methods).

Direct inguinal hernia is distinctly rare, especially so during childhood. Many surgeons consider the diagnosis of this condition unreliable unless it be confirmed at autopsy or at operation. **Femoral, lumbar, obturator, gluteal, sciatic, perineal, interstitial,** and **complete median hernias** may be encountered but they are all of very rare occurrence. **Diaphragmatic hernias** are occasionally experienced, together with transposition of the viscera. **Ventral hernias** are also found; usually these are situated in the median line between the ensiform cartilage and the umbilicus. As a rule they are small and contain only fat; they are due to minor defects in the rectus muscles.

Oblique inguinal hernia is very common and is much more frequently observed in boys than in girls. When a male child is the victim, there is a herniation into the funiculovaginal process or

more commonly into the retrofunicular space; but in the case of a female the hernia passes down into the canal of Nuck and produces tumor in the labia major. The sac of the hernia is usually preformed although the size of the inguinal ring and the elasticity and softness of the tissues here contribute to the ease of visceral herniation at this point. Smaller hernias usually contain omentum only, but those which are large contain one or more loops of intestine as well. Under these circumstances, the sac and its contents pass down the inguinal canal and fill one side of the scrotum. In approximately 4 per cent of cases the appendix is found, as well as intestinal loops; and occasionally Meckel's diverticulum is part of the contents of a hernia.

When the mass of the hernia has extended into the scrotum in an infant, there is but one condition with which it may be confused—a hydrocele. The hydrocele may be one either of the cord itself or of the processus vaginalis. The hydrocele, containing only fluid, will transmit light much more readily than a hernia. These facts, together with the knowledge that a hydrocele is irreducible, render the task of differential diagnosis one of comparative simplicity.

The *treatment* of hernias in infants is usually mechanical; reduction is often simple. According to Ochsner, 95 per cent of the hernias which occur in the first year result in spontaneous or nonoperative cure. In early infancy, mechanical prevention of the herniation is best accomplished by means of a wool truss. This is a skein of wool passed around the infant's body, drawn under the thighs, and so knotted as to bring pressure to bear over the inguinal rings. This wool truss has the advantages that it does not traumatize the tissues and that it is readily cleaned. (See Methods.) It is important that the child's nutrition should be kept in the best possible condition while retention by truss is being maintained, so that the advantage of healthy growing tissues may be added to the mechanical maneuvers and hasten the cure.

When incarceration of a hernia occurs, it is desirable to reduce the mass by manual manipulation if possible. In attempting this maneuver, utmost gentleness must be employed, and it is unwise ever to use manual manipulation when the strangulation is more than a few hours old.

To reduce a strangulated hernia by taxis, the child should be put into a hot bath to reduce muscular spasm. Immediately

after the bath, the patient should be placed on his back with the hips higher than the shoulders and the integument over the ring gently grasped with the fingers of one hand, while the operator attempts by a gentle enfolding movement of the thumb and fingers of the other hand to reduce the contents of the sac. If the maneuver is successful, there is usually a gurgling sound as the gut drops from the sac back into the abdomen.

The simple process of inverting the infant is sometimes of value. By this method, gravity traction on the gut and omentum is made to aid in the reduction. The child is grasped by the thighs as it lies on its back, and its body, with the exception of the back of the head and the shoulders, is lifted off the table. In the infant, the protective reflex of abdominal muscular contraction is not excited to the extent seen in the adult, and the inversion of the body, if gently done, does not cause the increased contraction of the ring that might be expected.

Operative treatment is required when there is such abnormal attachment of the muscles that an ultimate spontaneous cure cannot be expected; operation is also needed in those cases in which there is great difficulty in reduction and a tendency to strangulation. Similarly, should there be a hydrocele of the cord extending far into the inguinal canal, or an attached omentum, or if the canal be unusually wide and complicated by an undescended testis, operation is indicated. Strangulation, naturally, is an indication for immediate surgical intervention. These cases, as before noted, constitute only from 5 per cent to 7 per cent of the total number of hernias encountered.

Strangulated hernias are sometimes the causes of bowel obstruction. Strangulation may come on shortly after birth, but it is more usual to find it at a later time. The subject of an intestinal obstruction always gives evidence that he is suffering. The infant while lying quietly will suddenly cry out and draw up his legs and turn pale. The symptoms pass off as the pain subsides. Early in the observation of a case, vomiting will be noted. It is characteristic that this vomiting increases in intensity and becomes more frequent from hour to hour. In young infants, fecal vomiting is not long delayed. The child ceases to pass feces or flatus by the bowel, although one or two movements may come away in the earlier hours of the attack. These represent intestinal contents accumulated below the point of obstruction. After an enema has been given, the observer may be misled by the passage

of what is apparently flatus but which is in reality air that has been carried into the bowel along with the injected fluid. If an enema be ordered for a patient in whom a bowel obstruction is suspected, it is important that the nurse be instructed to exhaust all air from the tube of the irrigating outfit before she inserts the rectal tube.

Accumulation of flatus above the obstruction tends to produce an increasing distention of the abdomen. When such a child has become dehydrated, the contrast between the swollen abdomen and the shrunken limbs and thorax is very striking. The picture of abdominal shock—rapid pulse, extreme pallor, subnormal temperature and lethargy—quickly supervenes. If surgical intervention is not promptly undertaken and if the child survives the stage of abdominal shock, general peritonitis may take place. If this condition does occur, fever and distension of the abdomen will appear and generalized abdominal rigidity with tenderness, fever and sepsis will indicate the final stage.

Structures about the umbilicus are sometimes abnormal in form or position and contribute to an obstruction of the bowel. Most commonly the omphalic **diverticulum of Meckel** may form a bridge from the umbilicus to the mesentery, leaving a passage through which one or more loops of the small intestine herniate to produce a complete obstruction; or the bridge may entangle and incarcerate a knuckle of the gut in such a way that only a partial obstruction takes place.

The stump of the newly separated umbilical cord offers a portal of entry readily accessible to bacteria. The large amount of Wharton's jelly, in itself moist, provides a culture medium well suited to the organisms which produce **umbilical gangrene,** a condition in which the umbilical stump becomes oozing, greenish gray in color and of offensive odor. This type of infection rarely extends beyond the remnant of the cord and does not persist after the separation of the stump, although there may be some ulceration of the base of the cord and of the neighboring skin, which may become purulent and discharge pus for some time.

The name **omphalitis** is given the condition when the infection is severe, the tissues about the umbilicus are swollen, and much pus is produced. Omphalitis is usually complicated by a lymphangitis which spreads over the abdominal wall in lines radiating from the navel. The condition almost inevitably leads to a septicemia, and when it does it is accompanied by the usual sys-

temic evidences of that grave disorder. Occasionally the process localizes and abscesses of the abdominal wall develop. These are amenable to surgical treatment, and recovery may take place after incision and drainage; however, the more usual course is for peritonitis, and suppurative hepatitis with fatal pyemia to develop.

In very weakly babies infected at the umbilicus, spreading gangrene with extensive destruction of the abdominal wall can occur. Any of the vessels, either the arteries, the veins, or the lymphatics, may be involved in the inflammation; or the infection may cause a thrombosis without any apparent involvement of the other tissues about the navel. If there be a phlebitis, a subsequent fatal bacteremia is inevitable. This also may be the sequel when an umbilical artery is the subject of invasion, although sometimes, rather than a septicemia, a local abscess in the pelvis, or a peritonitis follows.

Peritonitis may occur in infancy either in an acute or a chronic form. The *chronic* forms of inflammation of the peritoneum, as they are found in early life are almost uniformly tuberculous in origin. The peculiarities, symptomatology and treatment of this form of peritonitis have been dealt with in this book in the paragraphs on tuberculosis in the chapter, "Infectious Diseases."

The *acute* form of peritonitis may be encountered at any time during infancy from the earliest days of life. In the newborn, implantations of bacteria about the umbilical stump usually provide a starting point for the infection. The offending bacterium is usually the streptococcus, and the peritonitis in the majority of cases is but a part of a general septicemia. Often it occurs subsequent to a hepatitis or a perihepatitis. Later in infancy, the rupture of an appendix vermiformis, traumatic perforation of the bowel, or the extension of infection after bowel obstruction, provides the commoner causes of peritonitis. Very rarely a gonococcal peritonitis has been reported as arising subsequent to a vulvovaginitis. More frequently, although still with rarity, a pneumococcus peritonitis has been encountered as part of a pneumococcus bacteremia or as an infection coming on with the appearance of a lobar pneumonia. An unusual subacute form of pneumococcus peritonitis sometimes occurs as a complication of neglected empyema, lung abscess or other foci of chronic pneumococcus infection.

The appearance of a patient suffering from acute peritonitis is always that of an extreme illness. The striking features are

high fever, vomiting, constipation, thirst, restlessness, rapid, thready pulse, and increased respiratory rate with inhibition of abdominal breathing. The facies are characteristic of abdominal shock—sunken eyes, drawn features and grayish pallor. In the beginning, the abdomen may be retracted, rigid and immobile; soon, however, abdominal distention develops with marked tympany. As the ballooning of the bowel progresses, it becomes impossible to determine the liver dullness or in fact to determine anything about the abdominal contents by palpation or percussion, although evidence of free fluid accumulating in the peritoneum may be elicited. The evidence of pain is positive; tenderness is extreme, and the rigidity of the overlying muscles is great. The child invariably lies on its back and it attempts to protect its abdomen by flexing the thighs on the body; however, in some instances older infants prefer the lateral position.

A striking and misleading feature of the peritonitis which follows the rupture of an appendix arises from the fact that a child who has been suffering from the colic and pain of an appendicitis has a period of some hours of entire relief after the rupture; and it is not unusual for parents to interpret the cessation of pain and the onset of comfort as an improvement of the condition. Not until 8 or 12 hours later do the first signs of general peritonitis become sufficiently positive to convince the untrained observer. Therefore it is a good rule for the attending physician to impose upon himself the immediate examination of a child under such circumstances. Whenever complaint has been made of persistent abdominal pain with vomiting, and this complaint is followed by a later message that the child is so much better that it does not need medical attention, the physician should insist on making a visit and examining the patient's abdomen. In this way a ruptured appendix abscess may sometimes be diagnosed and the patient relieved before the peritonitis becomes extensive.

In the earlier months of life, the abdominal symptoms of peritonitis may be so slight as to be entirely misleading. The most experienced clinicians have frequently been surprised to find an unsuspected suppurative peritonitis in the abdominal cavity of an infant at autopsy. It is possible for a very young baby to have an extensive inflammation in the peritoneum and to carry a considerable quantity of pus while the abdominal wall shows neither rigidity nor guarding and while there is no ascertainable evidence of

pain or tenderness. With this lack of abdominal disturbance, the distention of the bowel usual in older infants may also be wanting; as a result there is little or no respiratory embarrassment and the normal type of infantile abdominal breathing is hardly disturbed. The peritonitis of the newborn is almost invariably fatal, and acute peritonitis, of whatever origin, occurring during the first years of life allows little hope for recovery. Those forms of inflammation which follow rupture of the appendix or obstruction of the bowel, offer the most favorable prognosis, provided early incision and drainage have been possible.

The *treatment* of the condition is essentially surgical, but certain of the symptoms demand medical consideration and treatment. The child's comfort should be insured by placing it in an appropriate position in bed. A bed-cradle should carry the weight of the bed covers. Ingestion of food or fluid by mouth should be forbidden during the acute stage of the disease. Needed fluid should be provided by hypodermoclysis and the intravenous injections of hypertonic glucose solutions. If proctoclysis is tolerated without great pain, the rectal route for the administration of fluid may also be used. Great care must be taken not to purge the child; evacuation should be promoted by low enemas of simple salt solution. The vomiting, which so often is a troublesome symptom, is met best by stomach lavage.

The use of opium is contraindicated. In attempts to relieve pain, chloral is the most useful drug available; it should be given in doses of 3 grains for a 25-pound child, repeated once or twice at 4 hour intervals. When shock is evident, the application of heat in the form of hot packs and hot water bottles may be valuable. The intravenous injections of hypertonic glucose solution will also tend to combat the shock; and to these injections, adrenalin chloride may be added in 8 to 10 minim doses for a 25-pound child. If the glucose solution is made hot, the heat contributes to overcome shock. Certain French authors have reported that the use of saline solution injected at high temperature (up to 150° F.) has been a life-saving measure in the treatment of surgical shock. Intramuscular injections of camphor, 10 per cent in olive oil, are indicated; a 25-pound child who is threatened with collapse may have as much as 15 minims of this solution every 2 or 3 hours for 5 or 6 doses.

Infants who become emaciated as the result of malnutrition or rickets and some rachitic infants who are not particularly emaciated suffer from **prolapse of the rectum.** Prolapse occurs because

the tone of the pelvic muscles is lost and because the fat pads of the ischiorectal fossae disappear and the support which they give to the rectum is lost. This results in a relative redundancy of the rectal tube and permits its extrusion. Occasionally the bowel is truly redundant and the excess may be forced downward in attempts at stool.

Another factor which contributes to the prolapse is excessive straining whether at defecation or during micturition. Constipation, in which hard fecal masses come down into the anal canal to irritate it, is by far the most usual cause of straining. When constipation is a factor, the child should be given a teaspoonful of heavy petroleum oil 2 or 3 times daily; until the feces become soft the injection of 2 or 3 ounces of warm mineral oil into the rectum morning and night tends to allay irritation.

Diarrhea, which brings down acid feces from the upper bowel, may set up a proctitis, a distressing and ever present source of discomfort. The natural reaction to this discomfort is frequent effort at evacuation which aggravates the prolapse. In the presence of such diarrhea, accompanied by a prolapse, the indication is to wash the bowel once or twice daily with a small amount of alkaline solution. This should be followed by an injection of an ounce or two of ½ per cent solution of aluminum acetate.

Careful search must be made for polypi, tumors, fissures and thrombosed small hemorrhoids which may be keeping up the irritation and causing the frequent expulsive efforts. In a boy, narrowness of the urinary meatus and defects of the urethra, very rarely the presence of a stone in the bladder, may be contributory. More often, however, local irritative conditions in the rectum are sources of trouble.

The child should be taught to pass its bowel movements in the recumbent position while lying on its back; although if the condition is an aggravated one, the lateral position may be preferable. The evacuation should be made under the observation of an attendant who should be taught to return the bowel as it begins to prolapse, in order to prevent its extensive extrusion. The attendant should be instructed to grasp the prolapse loosely as it appears, hold it between the four fingers and the thumb surrounding its periphery and to make no immediate attempt to push the gut backward until the grasping thumb and fingers have squeezed the extruded mass gently from the periphery toward the center. This

maneuver is intended to empty the blood from the engorged vessels. Oftentimes, without any force whatever, the bowel may be returned; however, should it remain down after 30 or 40 seconds' pressure, the grasping hand pushes it upward and backward in a direction toward the middle of the sacrum. In the beginning of the treatment it may be necessary to return the bowel as many as 6 or 7 times during the act of defecation. The same maneuver may be used if the bowel protrudes between attempts at stool. A broad band of zinc oxide plaster should be applied across the buttocks in order to reinforce the weakened retaining power of the sphincter.

Unless the general condition of the patient is bettered, no amount of local treatment will bring about an amelioration. The malnutrition must be met by appropriate dietetic measures; if the child be rachitic, the use of an adequate diet together with the exhibition of cod-liver oil and phosphorus and the provision of sunlight and hygienic surroundings will be essential. All measures that tend to augment the weight of the baby and to increase the tone of its muscles are of value.

Surgical measures have been devised and recommended for the relief of this condition, but they are neither necessary nor advisable. Injections of hard paraffin into the ischiorectal fossæ have been made in order to replace the lost fat and acquire support for the bowel. Such injections are not without danger and, like other surgical procedures, are inadvisable in the treatment of a prolapsed rectum.

Abscesses, perirectal or **perianal,** may appear in the tissues around the rectum. As a rule these follow infections that have been made possible by abrasions, fissures and slight superficial ulcerations of the rectal mucous membrane. Usually they are painless, although sometimes before they develop fully they cause much distress. They may be superficial and be confined to the perianal tissues or they may be deep and run into the ischiorectal fossa. These abscesses are almost always accompanied by a high degree of fever. Sometimes a mysterious pyrexia will be explained by the discovery of pus around the rectum. Surgical incision is usually followed by prompt healing; therefore, it should be undertaken as soon as the condition is clear.

A neglected perianal abscess may lead to a **fistula in ano.** In infants these fistulas are for the most part confined to the superficial tissues, a fact which renders them much more amenable to treatment than the deeper lying fistulas which occur in adult life.

They are rarely complex; they take a straight course directly from the external opening to an internal opening which comes out at a point in the anal canal between the two layers of the sphincter. It is rare that they encircle the bowel to reach an inner opening in the midline posteriorly, a course so common when the affection takes place in the tissues of adults. As a result of their simplicity, the *treatment* is easy. A grooved director may be passed, the overlying tissues split and the fistula converted into an open wound. If this be packed for a few days with sterile or medicated gauze, granulation takes place and healing ensues without incident.

Very rarely pus finds its way deep into the ischiorectal fossa with the production of a true **ischiorectal abscess.** The appearances of such an abscess in an infant are identical with those seen in older patients. The tissues overlying the ischiorectal space become swollen, infiltrated and hard. The skin becomes reddened and shining and is tender to the touch, while the patient develops fever, rapid pulse and shows evidence of great pain. This form of abscess is much less frequently encountered in infancy as a result of trauma and infection of the rectum and anal canal than is the more superficial perianal abscess.

The pus from a *suppurative epiphysitis* arising anywhere about the pelvis may find its way into the ischiorectal fossa to produce an abscess there. In the earlier months of infancy, such a manifestation will probably be due to a pneumococcus epiphysitis. Later on, after the end of the first year, it is possible for a tuberculous infection to be the morbid process responsible. Confronted with an ischiorectal abscess without evidence of any rectal infection or inflammation, the pelvic bones and articulations should be carefully scrutinized for pathologic changes. Incision and evacuation of the abscess is the indicated method of treatment.

The **liver** is one of the important organs of metabolism during intrauterine and early infant life. Before birth, the liver is part of the hematopoietic system producing new red corpuscles. The hepatic production and storage of glycogen are functions which appear early in antenatal life, even so soon as the 5th month after conception; and the biliary secretions manifest themselves even earlier, often by the twelfth or fourteenth week of intrauterine existence. The great relative size of the infant liver indicates its importance at that age.

Normally, the infant's liver is palpable 1 to 1½ inches below the costal margin on the right side. The lax abdominal muscles with

the tendency to diastasis of the rectus muscles may permit a downward displacement or a rotation on the transverse axis so that apparent enlargement of the organ may appear, entirely unrelated to any pathologic process. True **hepatic displacements** may follow pleural effusions on the right side; sometimes they result from a pericardial effusion. As a very rare event during infancy, a poliomyelitis may leave the abdominal muscles so damaged that they are without any tone. Under such circumstances, especially if the right side of the abdomen is affected, the liver may descend so far that its lower margin lies below the level of the umbilicus.

Syphilis, rickets, and in later infancy the chronic forms of tuberculosis may any of them be the cause of a true **enlargement of the liver.** Engorgement from chronic failure of the circulation is less apt to cause great enlargement in a baby's liver than it is in the liver of an adult. Chronicity of lesion is a factor, as extreme cardiac decompensation is almost uniformly promptly fatal during infancy. The commonest cause of enlargement of the liver at this age is **fatty infiltration.** In contradistinction to the morbid process in fatty degeneration, which is uncommon during infancy, there is enlargement of the liver with no loss of true hepatic tissue but a deposition of fat within the liver cells. Fatty infiltration is essentially the result of a metabolic disturbance and it is found accompanying rickets, tuberculosis, syphilis, chronic indigestion and in obese children who receive an excessive carbohydrate diet.

In the greater number of cases of liver disease during infancy, **jaundice** is a pronounced symptom. It is encountered in the icterus of the newborn, in congenital obliteration of the bile ducts, in certain portal pyemias which follow umbilical infection and in those rare epidemic infections accompanied by jaundice which have been described, some cases as Winkel's disease and others as the malady of Buhl.

The commonest form of icterus as it occurs in later life, *acute catarrhal jaundice,* is not at all infrequent during the first two years of life and it may come on shortly after birth. The obstructive forms of jaundice, always excepting congenital obliteration of the bile ducts and the catarrhal form of jaundice, are practically unknown in infancy. Very rarely, marked yellowness of the skin will be encountered as a result of congenital syphilis; occasionally it occurs as the index of a cirrhosis of the liver. With a little more frequency acute and subacute hepatic atrophy are met, and most

infrequently an infant may be jaundiced because it is one of a related group suffering from **acholuric familial icterus.**

A very large proportion of newborn infants show the yellow stained skin of **icterus neonatorum.** Certain authorities believe every newborn baby to be in some degree icteric. The work of Lucas at the University of California Hospital has demonstrated the presence of an excess of bile in the blood serum of all newborn infants tested. However, not more than about 40 per cent of them are appreciably icteric; when they are, the yellowing is first apparent on the face and in the sclerotics. The tint may vary from the faintest yellow to a deep golden orange color; in the case of a jaundiced child who is also cyanotic, the color will be a deep bronze. This bronzing is also seen in cases of epidemic hemoglobinuria of the newborn in which cyanosis and icterus combine to a marked degree. In the milder cases of icterus neonatorum the urine does not respond to the tests for bile pigment, but bilirubin crystals may appear on the diapers; as the condition clears, the napkins may be found deeply bile-stained. The jaundice in this condition is rarely persistent; it becomes evident within 24 to 48 hours after birth and lasts only from 10 days to 2 weeks, although Still has reported cases of jaundice which seem to be of this type that have persisted for two months or more. When the diagnosis is certain, an icterus neonatorum need cause no alarm as it is probably the result, not of a disease process, but of an overdemand on the secretory cells of the liver. The bile is produced faster than it can be excreted and thus it accumulates in the blood. Besides the yellowing of the skin, the only other symptom of moment is the tendency to drowsiness and this is not extreme. The pulse may be slow but the slowing is not often sufficient to attract attention. It is characteristic of the malady that the stools carry sufficient bile to present the golden yellow color normal to infant evacuations. This point is of importance when the need to differentiate an icterus neonatorum from the jaundice which follows congenital obliteration of the bile ducts arises. It is also characteristic of the jaundice in congenital obliteration that it may appear some days, perhaps one or two weeks after birth. Unlike the decreasing simple jaundice of the newborn, the icterus of this condition increases in intensity as time goes on. The liver and spleen in a patient with icterus neonatorum are rarely enlarged, but they are almost uniformly increased in size when the yellow skin arises from bile duct obliteration.

It may sometimes become necessary to determine whether or not the icterus appearing shortly after birth is the evidence of *sepsis*. If this is the case, the child is almost certain to be appreciably ill. Usually there will be fever, although a weakly baby may have a marked degree of sepsis without pyrexia. Occasionally instead of a very acute, rapidly fatal bacteriemia or portal pyemia with multiple hepatic abscesses, the jaundice may be the result of a less acute infective process. An ascending inflammation may pass up the umbilical vein obliterating a portion of it to form a fibrous band which will mechanically obstruct the biliary duct.

Sometimes *syphilis* is the source of a *cirrhosis of the liver* the symptoms of which may appear shortly after birth, and among other evidences an extreme jaundice may be one. Jaundice may also be seen in those infrequent instances of inflammation and fibrosis of the bile ducts which follow invasion of the duct walls by the Spirochete pallida.

Congenital obliteration of the bile ducts is a rare occurrence in infant life, but it happens frequently enough to have 1 or 2 cases enter the clinical experience of most practitioners. It is the result of a congenital defect in the structure of the bile ducts which may have undergone a complete atresia or an extreme stenosis. The revealing symptom of the affection is jaundice which in most cases does not appear for 2 or 3 weeks, although it has been reported as well marked at birth. In the beginning the jaundice is slight but it becomes progressively deeper. It may vary from time to time but it never disappears entirely. The earlier stools passed, usually have the normal appearance of meconium but later on they become light without any trace of biliary coloring. The urine always contains bile pigment; the liver is large, hard and smooth, and splenomegaly is the rule. In many cases nutrition is well maintained for some months, but it finally fails and in all the reported cases the patient has died either from an intercurrent disease or from cholemic toxemia with convulsions. In a certain proportion of the cases the toxic features appear earlier and are more fulminant. Infants with this form of the disease may die in a few days with manifestations of hemorrhagic disease which is not amenable to treatment with whole blood or any of the measures usually effective in treating hemorrhagic disease of the newborn. It is especially in these cases that intractable oozing from the umbilicus occurs and large hemorrhages into the gastrointestinal tract take place. There is no record that a child with this disease has

survived for longer than a year and there is no treatment that holds out any promise of increasing the expectancy of life.

Simple **catarrhal icterus** is a disease quite common during infancy. It is probably of infective origin and it is known to have occurred in small local epidemics. It is an obstructive jaundice, the obstruction resulting from an inflammation about the ampulla of Vater. The swelling of the mucous membrane at the outlet of the duct is a direct cause of the obstruction; in many cases this inflammation is subsequent to a duodenitis. The appearance of jaundice is often delayed until 2 or 3 days after the beginning of the sickness. Malaise, irritability and restlessness are prodromal symptoms and after a few hours fever becomes evident. Vomiting usually occurs in young infants but it may or may not be a feature in the case of older babies. Often constipation is marked, although sometimes diarrhea is persistent and difficult to deal with. The stools are pale and, especially when the child is being fed on a milk diet, they are foul with the stench of fatty acids. Before the jaundice appears, the liver is enlarged although not to any great degree. It will be found tender to palpation and in children old enough to complain there may be spontaneous pain referred to the upper right quadrant of the abdomen.

With the development of the jaundice, abdominal pain and tenderness disappear, the liver volume progressively diminishes to normal proportions and the fever abates, while the urine becomes concentrated and darker in color, due to the excretion of bile pigment. The jaundice is first noticeable in the sclerotics and then about the face and finally spreads over the body. It varies in color from the slightest tinge of yellow to deep golden bronze; usually it begins to fade within 3 or 4 days after its appearance, and the fading is completed by the end of 2 or 3 weeks. Constipation tends to persist together with irritability and peevishness of the patient until toward the end of the attack. If the fever persists and cerebral manifestations develop and if petechial hemorrhages appear in the course of what seems an ordinary catarrhal duodenitis with icterus, these findings should lead the observer to suspect the presence of acute yellow atrophy or of some toxic source of the jaundice. In very young infants, it must be remembered that the appearance of the jaundice that accompanies congenital stenosis of the bile ducts may be delayed for 3 or 4 weeks; therefore, when a jaundice which has supervened at this time and which has been thought to have been of a simple catarrhal nature continues to in-

crease in intensity or if it fails to disappear after 2 or 3 weeks, the possibility that there is a congenital obstruction must not be overlooked.

The *treatment* of catarrhal jaundice resolves itself into a dietetic and hygienic management and the use of certain drugs given to relieve annoying symptoms. The condition is self-limited, but it is well demonstrated that the underlying duodenitis is aggravated by foods which contain high concentrations of fat or of fatty acids and also the coarse vegetables, whole wheat and bran breads and whole meal porridges.

During the first 24 hours, the vomiting is best treated by the limitation of the intake to water, either plain or alkaline. The use of an alkaline purgative may be of advantage; of these, none is better than milk of magnesia in doses varying from 1 to 6 teaspoonfuls according to the age and weight of the patient. It is best given in divided doses, well iced, a teaspoonful or less at ½ hour intervals until the required amount is taken. For older infants, in addition to the milk of magnesia, syrup of rhubarb may be used with advantage. Castor oil and calomel are to be avoided because they are irritant and both may aggravate the vomiting and increase the inflammatory process in the duodenum. When a more drastic purge than the milk of magnesia is needed, a combination of sodium sulphate grains 5, sodium bicarbonate grains 3, sodium salicylate grains 3, may be given with ½ dram each of syrup of rhubarb and simple elixir every hour for 5 or 6 doses. If the vomiting is persistent and troublesome, the stomach should be thoroughly washed out and 6 or 8 ounces of a 2 per cent bicarbonate of soda solution be poured through the tube and left in the stomach.

After the vomiting has subsided, bottle-fed infants should be given formulas of whey or skimmed milk with cereal gruel. The cereals used should be fine in form and thoroughly cooked. Lactose or maltose may be used to reinforce the feeding, but at least in the early days of this disorder it is well to omit them. Older infants in the later stages of the treatment may be given broths thickened with cornstarch, arrow-root, sago or the fine Italian pastes such as pastine or vermicelli; still later, scraped meat, egg-white and skimmed milk may be added. Children who have once had an attack of catarrhal jaundice are apt to suffer recurrences; therefore, care should be taken to see that between attacks they are not overfed with fat or with coarse, irritating foods. The onset of some cases of recurrent vomiting can be traced to an initial attack

of catarrhal jaundice. The child under treatment for this form of jaundice should be kept quiet in bed and be protected from cold, but at the same time he should not be overclothed. As in any other illness, fresh air and sunshine are indicated as valuable hygienic aids to treatment.

Acute or subacute **yellow atrophy of the liver** may appear at any time during childhood. In infancy the acute, rapidly progressive form, while exceedingly rare, is the one met more often than the insidious, subacute type. Ernest Dickson has studied and recorded the pathological findings in the hepatic tissues of a fatal case of *subacute* atrophy that occurred in the writers' practice. It is generally stated that syphilis is a factor of influence in these atrophies of the liver; however, in this case it was definitely possible to exclude syphilis. This patient's jaundice appeared during the second week of life and the child died in its fifth month. A striking feature was that the tissue turgor and muscle tone were good in spite of the high degree of jaundice, until shortly before the fatal issue. The child weighed 8 pounds at birth, was breast-fed for 2 months and then was put on a normal diluted milk and lactose formula upon which he thrived. Towards the end of the second week of his life he became deeply jaundiced. This was the only sign of disturbance; there was no gastrointestinal upset nor evidence of any other illness and the stools were persistently yellow. When the child had reached its third month he seemed healthy and happy and besides the jaundice and a palpably enlarged spleen there were no striking findings. For 6 weeks he continued in the same way, the jaundice increasing in intensity, with a steady gain in weight at the same time. At the end of the fifth month of life there was a sudden onset of respiratory distress with marked abdominal distention. Drowsiness and lethargy appeared and the child passed into coma and died. Autopsy revealed marked ascites, a small granular liver, a large indurated spleen and large kidneys which showed parenchymatous degeneration. There was no stenosis of the bile ducts. The microscope showed a complete necrosis of the whole of the liver except a narrow zone around the margin where there was evidence of regeneration of the secreting tissue.

This case is of especial interest because it illustrates that life is possible in the presence of a very small amount of functioning liver tissue and that extensive destruction of the liver may take

place without the disappearance of biliary secretion or the development of hepatic insufficiency.

The more common form, the **acute yellow atrophy of the liver,** is first made manifest by a jaundice in no way differentiable from that of the ordinary acute catarrhal icterus. There is depression, constipation, vomiting and slight fever—a clinical picture which might well form part of the lesser disease. However, these symptoms instead of abating and disappearing grow more profound with the passage of time; the skin becomes yellower, and the seizures of vomiting are more frequent and of greater severity. As a rule the temperature rises although subnormal temperatures sometimes accompany the later stages of the disease. Blood appears in the vomitus, in the stools and under the skin. The central nervous system shows involvement, and twitchings, isolated muscular spasms and convulsions occur. In older infants there may be marked delirium between convulsions, or the excitability may be replaced by drowsiness and lethargy. Such a somnolence always marks the final stages of the disease and invariably the patient goes into a coma preceding death.

In the beginning of the attack, the liver is markedly enlarged and this finding, together with a profound icterus, should put the attending physician on guard, for simple catarrhal jaundice is rarely accompanied by so great an enlargement of the liver. After a few days, however, there is a rapid diminution in the hepatic volume; so much so that late in the course of the disease it may be impossible to demonstrate any area of liver dullness. Throughout the illness the spleen usually remains large. The urine is characteristic, diminished in volume, dark in color, and it carries much bile pigment, albumen and casts, which are always bile-stained. The output of urea is always lessened; leucin and tyrosin also are to be found; these are pathognomonic of this particular form of liver degeneration. The greater number of cases of acute and subacute atrophy terminate fatally within from 10 to 20 days. There are certain cases which have been reported by good observers where the stage of pyrexia, hemorrhage and cerebral involvement gave way slowly to a period of defervescence which resulted in the final recovery of the infant. Such a happy outcome, however, is too rare to be expected.

Certain cases of icterus appearing shortly after birth may clear up, only to recur later. These may fall into the category of a **congenital family jaundice (familial cholemia).** Splenomegaly and hepatic enlargement are characteristic of this form of jaun-

dice. Uniformly there is present an anemia which very often is profound enough to cause death soon after the child is born. Beside a great reduction in the number of red cells, the blood shows the presence of normoblasts and megaloblasts. The urine is free from bile but carries some urobilin and usually traces of albumen. The stools are not acholic but present a normal appearance. An inquiry into the family history will reveal that jaundice has appeared in many of its members. A certain proportion of infants who show the evidences of this disease at birth die; but if they survive the severe anemia and live beyond the end of the first month, they will probably attain adult life with a fair degree of health and no other inconvenience than recurrent attacks of jaundice and anemia. Such infants are susceptible to chill and are naturally of lower resistance than normal infants and are more likely to die from diarrheas and respiratory complications.

The *treatment* is purely dietetic and hygienic. The maintenance of warmth must be assured in sunny, well ventilated surroundings. Carefully regulated diet is of importance, baths and massage may contribute something to the well being of the child but no other known therapeutic measures have any influence on the course of the disease.

Cirrhosis of the liver may occur during the first 2 years of life. Portal cirrhosis, the ordinary gin drinker's liver, is almost unknown in America; it is occasionally seen in European communities where wine and diluted spirits are considered appropriate additions to the infant's diet. The appearance of a child with such an affection varies with the stage of the disease, but the outcome is almost always the death of the patient. The earlier appearances are those of a gradually developing malnutrition, and late in the disease jaundice and abdominal enlargement from ascites occur. Early in the disorder an enlarged liver and spleen are usual manifestations, while later hepatic atrophy and dilatation of the superficial abdominal veins are common. Such a picture is hardly likely to develop before the second year of life; when it does the clinical aspect is such that the condition may be readily mistaken for a peritoneal tuberculosis.

Biliary cirrhosis, sometimes known as **hypertrophic cirrhosis** or **Hanot's cirrhosis**, is never seen in infancy although Mexican children, even babies, suffer from a form of interlobular cirrhosis in which the pathological appearances of the liver are very like the findings of an ordinary biliary cirrhosis. Hanot's cirrhosis as it

occurs in older children is characterized by an extended course during which recurrent crises, each lasting a few days, supervene. These exacerbations of the disease are followed by an increasing enlargement of the liver. The disease as seen in Mexico is very acute; the course is short and recurrences are infrequent because as a rule the patients die when the disease has lasted but 2 or 3 months. Enlargement of the spleen which is always a feature of the ordinary hypertrophic cirrhosis does not accompany this special form of cirrhosis; while ascites which is rarely seen in the form of the disease described by Hanot is practically always a complication or at least a terminal event of interlobular cirrhosis encountered amongst Mexican babies.

An involvement of the liver is an inevitable occurrence in the course of *congenital syphilis*. The signs of liver involvement appear during early infancy, often within the first month of life. There may be a mild jaundice although this manifestation is far from constant. The snuffles, nutritive impairment, anemia and rash are more frequently seen; but even these signs of syphilis may fail and there may be nothing further to reveal the presence of the disease than the enlargement of the liver and spleen, both of which will feel hard and smooth to the palpating hand. In all cases in which enlargement of the spleen and liver is encountered during early infancy, whether or not these enlargements be accompanied by other evidences of syphilis, Wassermann tests should be made. Antisyphilitic treatment, instituted early is almost invariably followed by a recession in the size of the liver and an improvement of the general condition of the patient except in those cases in which an ascites develops early.

Any **new growth** may appear in the liver. None of them are important contributors to the pathology of infancy except **sarcoma** which in rare instances may give rise to great enlargement of the liver, an enlargement which in turn may cause much distention of the abdomen. Palpation of the enlarged organ shows it to be rough and irregular in contour. As a result of the growth, jaundice may develop and ascites may follow. The growth may be primary but more often it is the result of metastasis from a tumor originating in the Wolffian tract, especially about the kidney or suprarenal region.

Of benign tumors, **angiomas** are the most common. These are of little importance save for the fact that on the rarest occasions

they have been known to rupture and cause the patient's death from hemorrhage into the peritoneum.

Multiple abscesses occur in the liver of infants as a result of the transportation of septic thrombi to the liver through its blood vessels. A child so affected exhibits septic pyrexia with vomiting, sweating and cyanosis. Jaundice may be a marked feature, or it may be entirely absent. The evidences of abdominal pain and tenderness are always present. In infancy the condition is rarely seen apart from a pyemia and it is seldom possible to differentiate it from the peritonitis which inevitably succeeds it. The condition is uniformly fatal in spite of any form of *treatment* that can be devised.

Disorders and disturbances of the pancreas play but an unimportant part in the diseases of infancy.

The **pancreatic functions** of the infant are the same as those of the adult. The ferments contained in the secretions of the gland are protease, amylase and lipase. In **functional diseases of the pancreas** as they occur during childhood these ferments may be affected. Lipase is most frequently the ferment which suffers diminution in its activity. As a result of this disturbance, unsplit fats appear in the stools in greater or less quantity. In the more severe affections of the pancreas, protease may be diminished or absent, with the consequence that proteins, especially meat fibers, escape digestion and appear in the stools. In extreme degrees of pancreatic insufficiency, the secretion of amylase fails. When the secretions of all three ferments are interfered with, starches as well as proteins and unsplit fats, together with inspissated intestinal secretions compose the bulk of the stools.

These alterations in food-splitting power are indices of the condition known as **pancreatic insufficiency** or **celiac disease**, a morbid state encountered with no great frequency during infancy. It may be the result of damage to the pancreas from the influence of absorbed toxins, although it does occur as a sequel of an ascending infection of the pancreatic ducts. The toxins most often responsible for the alterations in pancreatic power are those absorbed from the intestinal tract when the latter is inhabited by a deleterious bacterial flora. Less frequently, syphilis or tuberculosis may be the causative pathological process, but the influence of syphilis leads more frequently to fibrosis and chronic pancreatitis.

In some cases in which pancreatic insufficiency is a feature, the

elaboration, not alone of the external secretions but of the internal as well, is interfered with. Under these circumstances, together with the incomplete digestion of food, there are evidences of marked infantilism, a state of affairs well described by Byron Bramwell as **pancreatic infantilism.** It is possible that the cases first discussed by Herter as cases of **intestinal infantilism** also belong in this category. There is every reason to believe that some of the fat indigestions of infancy are due to a mild insufficiency or inefficiency of pancreatic lipase.

Acute pancreatitis is a disease that is almost never seen during the first 2 years of life. Most often in childhood it is a complication of epidemic parotitis although it has been recorded as an accompaniment of the acute infectious diseases. When it appears as a part of mumps, the prognosis is good; the condition then clears up spontaneously after a few days. The clinical aspect that presents itself is that during the course of an attack of mumps abdominal pain with shock, together with vomiting and diarrhea, supervenes. Abdominal tenderness is extreme and fever is absent; the urine may contain sugar and the stools much unsplit fat. The *treatment* is directed toward alleviating the pain and supporting the patient. Hot compresses to the abdomen are applied and if necessary a dose of codein or morphin appropriate to the weight and age of the child may be given.

Chronic pancreatitis is even rarer than the acute form. It is usually but a manifestation of congenital syphilis. The signs of its presence are chiefly the evidences of unsplit fat in the stools of a syphilitic infant; the general treatment is that of the constitutional disease.

Involvement of the **pancreas** is not uncommon in the course of a generalized **tuberculosis** and the gland may be involved in a local process by extension from a tuberculous peritoneum.

Pancreatic stones and cysts of the pancreas are possible developments but so far are unrecorded in the pathological annals of infancy.

CHAPTER XIV

DISEASES OF THE HEART AND CIRCULATION

The mass of the heart of a newborn baby in relation to the body weight is greater than that of an adult. By the end of the second year the organ has doubled in weight. This rapid growth, with the accompanying adjustment of the circulatory system, accounts for some of the malnutrition and some of the disturbances of metabolism so frequent during the second year of life, events too often attributed to digestive disorders alone.

During early infancy, the right and the left ventricles differ little in their size and in the thickness of their walls; but in the course of a year or two the cavity of the right ventricle increases in size without any coincident thickening of the walls. At the same time, the wall of the left ventricle becomes thicker and more powerful in order to meet the increasing strain of the growing systemic circulation. During early life the caliber of the aorta and of the pulmonary arteries is proportionately larger, measured against the volume of the ventricles, than it is at any later period of life. In fact, a total cross section of the whole arterial system is relatively greater at this age, with the result that blood pressure is of necessity low. It is extremely difficult to make an accurate estimate of *blood pressure* in infancy, and this clinical method, so valuable an aid in a study of older children and of adults, is of little practical value in appraising cardiac and vascular diseases as they occur during infancy.

These peculiarities of the vascular system, render the *pulse rate* subject to rapid variations and make it a poor guide to the clinician. Early in life there is always a definite *respiratory irregularity* of the pulse to be found; frequently such an irregularity is exaggerated by a change in position. These variations are entirely physiologic. During sleep it is very common to find peculiar irregularities of the heart with grouped beats and unequal pauses. In the case of certain babies, and these are not necessarily infants who show disturbances of the nervous system, the presence of respiratory irregularity is evidenced by a slowing of the heart rate during full inspiration. Although they have no ill significance, such phenomena may cause undue concern for fear that the

child affected is subject to a cardiac disorder with respiratory embarrassment; a slowly beating heart may be perfectly normal and the slow pulse rate be a familial peculiarity.

The results of percussion and the position of the maximum cardiac impulse will vary materially according to the shape of the infant's chest. The large size of the right heart of a baby in relation to the left, together with the greater relative mass of the abdominal contents and the high position of the diaphragm, causes the heart to lie well up in the chest, higher than is usually the case in adult life. At this early age, the heart is rotated on its vertical axis in such a way that more of the right side is brought forward against the chest wall; thus the delimitable area of cardiac dullness is widened. In general, in the first two years of life, there is found a heart whose apical impulse lies in the fifth interspace or above and in the nipple line or just outside it. In healthy infants, the organ often extends as far as one-half to three-quarters of an inch to the right of the right sternal border and produces a dullness typical of the relatively large auricle and great vessels. This dullness may reach up high enough to blend with that of a normal thymus. This blending of cardiac and thymus dullness may make it difficult to determine whether or not we are dealing with a true enlargement of the thymus gland.

Heart block and premature contractions are extremely rare, but they have been reported in infancy as a result of toxic or of digestive disturbance.

A *bruit* heard over the heart of an infant does not of itself indicate cardiac disease. The greatest care should be taken to gather other evidences of heart involvement. Unless these be found, a conclusion as to the diagnosis of an actual lesion should be withheld and a decision delayed until after an interval of some days. Many a family has been needlessly alarmed and many an infant unduly restrained for no reason other than the presence over its cardiac region of a bruit, which was no more than the evidence of a mild structural abnormality of no physiologic consequence.

Even in earliest infancy, bruits may be encountered. Many of these cannot be related to any organic change in the heart, a fact which renders the evaluation of the *murmurs that occur in the absence of valvular deformities*, a matter of some difficulty. These sounds are by no means limited to the region of the base of the heart, nor do they occur only in anemic, marantic or rachitic infants; although in such children, it is the rule,

rather than the exception, to find a loud systolic bruit audible, sometimes at the base, sometimes at the apex, frequently over the entire precordium. It is characteristic of such murmurs that their transmission is not wide, but exceptions occur, especially when they are to be heard above the pulmonic area; these latter bruits are transmitted towards the subclavicular region and they are often audible when the upper left side of the back is auscultated. On the other hand, bruits due to organic diseases, except a few that result from septal deficiencies, are widely distributed and almost invariably they can be heard transmitted to the axilla and the various regions of the back.

The **cardiorespiratory murmur** is a bruit that is sometimes to be heard during the second year of life; it is often difficult to differentiate the noise produced by the air moving out of the neighboring alveoli under the impulse of the heart beat, from a true murmur produced in the moving blood. The cardiorespiratory murmur is usually heard on the left side of the chest toward the base of the heart in the second and third intercostal spaces, although sometimes it is audible at the right border. When present, **pericardial friction** is heard in the same area; therefore, some confusion may arise between the two conditions. However, the pericardial friction sound is to and fro in character, while the cardiorespiratory sound is largely systolic in time.

The bruit that follows slight degrees of **patency of the interventricular septum** is usually found at its maximum about the third costal cartilage inside the apex beat. It is superficial in character and it may be accompanied by a thrill. When a bruit is heard at the base of the heart, if it be functional, it is loudest about the pulmonary valve area. It varies with the patient's position, and it may be heard over the great veins, the subclavians and the jugulars. The **murmurs of aortic and pulmonary stenosis** show the same conduction. In **aortic stenosis** the left ventricle is hypertrophied, the pulse is small and there is a systolic thrill to be felt high up on the chest. When there is coarctation of the aorta above the level of the valve, aside from a basal systolic bruit transmitted to the neck, there may be no other sign of cardiac involvement. On the whole, a study of the heart bruits that may be heard during infancy tends to emphasize the wisdom of Stephen McKenzie's teaching that physical signs produced by the heart during infancy, unaccompanied by signs of circulatory failure, should give us little concern because they are rarely evidences of cardiac insufficiency.

In the first two years of life, **acute heart disease** is rare, even as a complication of sepsis or of pneumonia, although the pericardium may be involved by extension from a pneumococcus pleurisy. Rheumatism, the common source of **pericarditis** and **myocarditis** in later childhood, is rarely etiologic during babyhood. **Carditis,** as a *complication of scarlet fever,* shows no age preference and infants develop a myocarditis, or a pericarditis and endocarditis of a subacute nature which may result in adherent pericardium and in deformities of the heart valves.

Invasion by the pneumococcus is by far the most frequent cause of heart lesions during early infancy; most characteristic of the ravages of this organism is **suppurative pericarditis,** a rare accompaniment of empyema. Endocarditis and myocarditis of this origin are also met, but they rarely happen as complications of suppurative pericarditis; most often there is an accompanying empyema. Staphylococci are sometimes responsible for endocarditis in infancy. Frequently, the inflammation can be traced to infection from a primary suppurative focus such as an osteomyelitis, a furunculosis, an otitis media, a septic bronchitis or an infected wound. The streptococcal infections may be complicated by an endocarditis, by a pericarditis or by both. In such cases the upper respiratory tract is usually the source of the infection, and in many instances, blood cultures will reveal an accompanying streptococcal septicemia.

Acute endocarditis in infancy sometimes presents a puzzling diagnostic problem. The relative weakness of the cardiac muscle is such that dilatation occurs early in the course of the inflammation. The endocardium is so fine and the valve structures so tenuous that it may be impossible to hear bruits, especially in the earlier stages of the disease. This physical sign, so valuable as a diagnostic aid when we are dealing with older children or adults, is not nearly so helpful when we are studying an infant patient. In babyhood the endocarditis is often an accompaniment of a bacteriemia.

Later on, if the child survives, valve deformities may ensue, and as the dilatation diminishes, the heart muscle becomes more vigorous, and the blood, driven through deformed valve orifices, produces sounds analogous to those heard in more mature hearts. At this advanced stage of endocarditis in infancy, it is usually at the mitral valve that the bruits are produced and they are presystolic and systolic in time. Sometimes, even when there is a

very marked narrowing, the presystolic murmur is but slightly audible.

Occasionally the major incidence of the inflammation falls upon the aortic valves, and the child develops a true **aortic regurgitation** or a **combined aortic regurgitation and stenosis.** With this there may be a characteristic enlargement of the left side of the heart and a typical basal diastolic bruit, with a lowering and a widening of the maximum palpable cardiac impulse. Rarely, with aortic disease, there is a secondary damage to the mitral valves and, as a result of this, a later dilatation of the other side of the heart follows. The infantile characteristics of the myocardium are such that almost always when there is marked acquired valvular defect, dilatation of the cardiac chambers comes into evidence, and a later enlargement of the liver together with visible pulsation of the veins in the neck may be demonstrated.

It may be repeated that the inflammations in the endocardium resulting in valvular deformity are rare, much rarer even than pericarditis which itself, is not a common disorder.

The early detection of **pericarditis without effusion** is a very difficult matter when one is dealing with infants, for sometimes a developing pericarditis produces no audible sound, and at other times the sounds are barely discernible and are difficult to evaluate. Then too, it is a fact that much of the pericarditis of infancy occurs as a complication of respiratory disease, in which the signs of lung involvement obscure the early evidences of the pericarditis.

The pain of pleuropericardial inflammation may be the cause of much distress which the examiner may find difficult to relate to its proper origin. Cough and wasting, as well as vomiting and diarrhea, are symptoms of such common occurrence in the early years of life that although they are generally present in this condition, they have no distinct diagnostic value. In any event, when in the course of a pleurisy there is reason to suspect an involvement of the pericardium, frequent and painstaking search for friction sounds should be made with a stethoscope placed over the base of the heart, for it is here that pericardial friction is most apt to be heard. A little later, when **pericardial effusion** is taking place, stress can be laid upon the gradual muffling of the tones of the heart and on the development of an increasing area of dullness, together with a shifting of the maximum cardiac impulse from the left edge to well within the area of percussion dullness. When these physical signs appear, a radiogram should

be made, and it will be decisive in determining the presence of an effusion.

In neglected cases when the effusion is large, the pulse is small, easily compressible and usually rapid. Ventricular insufficiency permits a fullness in the veins of the neck, and there is some edema of the face, together with a distressing condition of air hunger. Under these conditions, the increase in the area of the cardiac dullness is upward and to the left; together with this change, there may be marked compression of the upper lobe of the left lung. In such circumstances, especially when the pericardial effusion is purulent, the child will be extremely ill, very pale, will suffer from paroxysmal attacks of dyspnea, irregular and extreme variations of temperature, and occasional seizures of syncope, and perhaps convulsions. Complicating empyema or unresolved pneumonia, such a clinical picture in an infant inevitably means the presence of a **purulent pericarditis.** Sudden death usually follows in the course of a sepsis or during a syncopic convulsion, although it may happen apart from these seizures, as a result of some slight exertion. As part of an inflammation of the pericardium, the chronic condition, **adherent pericardium,** may occur. If this condition develops very early in life, the embarrassment of the circulation which ensues leads to relatively rapid fatality.

The *treatment* of a pericarditis in its earlier stages calls for rest and the application of an ice bag to the precordium for the relief of pain. The ice bag should be suspended so that its weight is not borne by the chest of the infant, and it should be applied ½ an hour out of each 2 hours. Opium in the form of codein or of the deodorized tincture is of value, $\frac{1}{20}$ grain to a 25-pound child, or ½ minim of the tincture to a child of like weight at 8 to 12 hour intervals; not only because it aids in dulling pain is it helpful, but also because through its use we may enforce rest for the patient. Dietetic measures are to be carried out with care; the vigor of the digestive tract should be maintained and evacuation of the bowels promoted in order that accumulated flatus may not further embarrass the heart through pressure on the diaphragm.

If slight, the effusion of *serous* pericarditis will usually absorb spontaneously before great circulatory embarrassment develops; but in a case in which this course is not taken, it will become necessary to withdraw the fluid. The best site for pericardial puncture is in the middle line just below the xiphoid cartilage.

The needle is to be thrust through the skin at this point, turned and pushed through the diaphragm and pericardium while held parallel to the long axis of the body. It is wise not to undertake such a procedure until after a radiogram has been made and the presence of fluid in the pericardial sac confirmed. If the fluid withdrawn is found to be purulent, the probability that any surgical procedure will be of aid to the infant is not great; however, incision and drainage of the pericardium may be tried; success may crown the effort, if the case be one of the very few which occurs apart from a large focus of pus elsewhere.

The **toxins of diphtheria** produce profound changes both in the muscle and nervous apparatus of the heart. It is quite unusual, however, for diphtheria to be the cause of great heart involvement in children under two years of age. The same may be said about the effect of the toxins of measles, of influenza and of meningococcic infection during this period of life.

Damage to the myocardium as a result of these bacterial toxins may be made evident by an undue irregularity of the heart's action, together with a slowing of its rate. When the pulse is greatly slowed, vomiting and prostration with syncopic attacks may occur. These conditions may develop within the first week of the disease and in such case, a short, systolic, mitral murmur may be heard.

In contradistinction to these early oncoming cardiac failures, there is a type of heart weakness following diphtheritic infection, in which the appearance of symptoms is delayed for several weeks after the apparent cure of the disease. The evidences of heart failure occur coincidentally with palsies and with the loss of the deep reflexes. These postdiphtheritic pathologic conditions are rare among very young children; and when they do occur, they are apt to be mild and transitory. None the less, as the most profound myocardial damage may be present, it is necessary to observe with great care those infants who have been attacked by diphtheria, and to regulate their regimen in such a way as to protect them from undue exertion during the period of potential danger. Straining at stool is especially to be avoided and the desire of anxious parents to return a runabout infant to usual activity oversoon must be repressed. The prompt injection of a sufficient dosage of antitoxin in a case of diphtheria, through its prophylactic effect, is the most efficacious means of dealing with a possible myocarditis, although not even the best treatment will always prevent damage to the heart muscle.

Drugs are of little value in the treatment of toxic myocarditis, and they are often actually harmful because the myocardium is not in a condition to respond. Strychnine, so frequently recommended, is unquestionably of evil effect. It stimulates the sensorium of a child already restless, and it has no good effect on the cardiac muscle. When the heart is slowed, atropin may be of some use provided the degeneration of the heart muscle is not too great. Occasionally when cyanosis is deep and the red blood cells are not being aerated properly, oxygen inhalations are helpful; but when all is said and done, apart from antitoxin, the only therapeutic measure that can be of avail is the maintenance of absolute rest, and the only drug of much use is one that will insure quiet and immobility. For this purpose, opium is the indicated remedy. The use of enough codein or deodorized tincture of opium to keep the child just drowsy and to prevent the overwhelming irritability that causes it to move in continuous restlessness hour after hour, will often prove to be a life-saving measure. The dosage should be the minimum that will produce this effect. In a 25-pound child, $\frac{1}{20}$ grain of codein or $\frac{3}{4}$ minim of the deodorized tincture of opium every 6 or 8 hours should be given or withheld according to the judgment of the attending physician.

It is important both for treatment and prognosis to distinguish between the **congenital** and **acquired forms of cardiac disease** as it is met during the first two years of life. It is rarely difficult to arrive at an accurate diagnosis of the congenital character of the disorder. The early appearance of the signs and symptoms, the history of the case, especially when it is clear that the child has not suffered from an acute pneumococcus or streptococcus infection, the dominance of physical signs in the heart without the appearance of proportionate cardiac symptoms –these peculiarities found in the case of an infant testify that the pathologic conditions present in the heart are of congenital origin. Marked cyanosis may be present and when it is, this phenomenon is always most suggestive of congenital cardiac defect. If the cyanosis occurs in paroxysmal attacks, it is usually accompanied by seizures of dyspnea which are also paroxysmal. Such a clinical picture rarely happens in infancy apart from the manifestation of congenital cardiac disease; however confusion may arise in the early weeks of life when the child may be subject to recurrent cyanotic attacks due to partial atelectasis of the lung or to symptoms that accompany enlargement of the thymus.

The ability accurately to determine certain given cardiac anomalies may be of great value in helping to arrive at a valid prognosis. Much stress has been laid on coincident defects in other parts of the body. Though it is only natural when the potential of one part of the germ plasm is interfered with that other parts may exhibit the effects of a like disturbance; however, too much stress must not be laid on these defects as aids to diagnosis as the best authorities seem to find that they occur in only about 20 per cent of the cases.

Certain symptoms that occur during infancy are common to all cases of this group of congenital lesions, although not all the symptoms occur in every case. The most common and most striking symptomatic expression of such congenital cardiac defects is *dyspnea,* which is apt to be paroxysmal in type. The paroxysms may be extreme and are very alarming to see. Such attacks coming on during the first 5 or 6 months of life should lead to a searching examination of the heart for a congenital defect. The dyspnea may be accompanied by a cyanosis which is well developed during the attacks, and slight or absent between them. Not always, however, is the dyspnea paroxysmal; it may be constant and the child may be subject to short, rapid, panting, phased breathing. Such an infant will be very sensitive to cold and to heat, and great difficulty may be encountered in maintaining its nutrition. If the child survives the early months, a chronic malnutrition may ensue, or the result may be a cardiac infantilism. Occasionally, such a child as it grows older may present the evidence of mental deficiency, although it is hardly probable that this is the result of the circulatory failure, but rather the effect of a coincident anomaly of development. However, the failure of the cerebral circulation does have a very definite influence over the temperament and emotional stability of children, especially during their second year. Cardiopathic infants often exhibit mental irritability with fits of screaming and outbursts of ungovernable temper.

Cyanosis occurs as a result of congenital cardiac disease in about two-thirds of the cases. Sometimes it is hardly noticeable except on exertion. It is best seen during the attacks of angry crying so easily induced in these cardiopathic babies. Often the cyanosis is so slight that it may escape observation for the first few months of life, or be seen at this time only when the child is screaming or suffering from slight respiratory involvement that would have no effect on the color of a normal baby. Exposure to

cold and to unusual heat, especially the former, may aggravate a slight cyanosis and make it noticeable. Cyanosis, however, is no index of the gravity of the cardiac condition, for many patients who are exceedingly blue during their babyhood survive to useful adult lives. On the other hand, many children whose cyanosis is hardly visible during the first year of life, develop it to a high degree when they become runabouts and demand greater effort from the heart. Like dyspnea, cyanosis may be paroxysmal and this paroxysmal cyanosis occurring apart from dyspnea, has been stated to be pathognomonic of defects of the interventricular septum. However, cyanosis by no means always occurs when there is a septal defect.

Instead of cyanosis, extreme pallor may characterize the skin of an infant suffering with a congenital cardiac lesion, and when this symptom is present, whether it is developed at birth or comes on later, it is of evil prognosis.

To differentiate accurately the various types of congenital lesion is a matter of extreme difficulty unless the observer has had opportunity to see a great many cases of cardiac anomaly. In this as in all other clinical matters, experience is the touchstone which renders diagnosis and prognosis clearer. A history of the presence or absence of cyanosis may be of much assistance in arriving at a correct conclusion.

Abnormal communication between the aorta and the pulmonary artery, an **open ductus arteriosus** and **simple septal defects** are conditions which occur, usually without cyanosis, although there are cases on record in which all or any one of these anomalies has been accompanied by marked blueness. When a septal defect is extreme, as happens in the biloculate heart, or where the truncus arteriosus has failed to divide properly, or where the great vessels, aortic and pulmonary, are transposed, cyanosis may be of high degree. This excess of cyanosis almost without exception shows, when there is either an **atresia** or a **stenosis at the pulmonary outlet** of the heart. Extreme cyanosis without physical signs other than well marked accentuation of the second pulmonary sound suggests **complete transposition of the great vessels.**

The degree and character of dyspnea is also sometimes of aid in helping to differentiate the various forms of congenital cardiac disorders. Without cyanosis, or with mild seizures of blueness, dyspnea in paroxysmal attacks often marks the presence of a patent ductus arteriosus or of a persistent **foramen ovale.** These

attacks often begin shortly after birth, and are then very alarming in appearance. On the other hand, the first appearance of the seizure is often delayed until the child is some weeks of age. Such a delay complicates the problem of diagnosis for the observer. This delay in the appearance of paroxysmal cyanosis and dyspnea is probably due to a late closure either of a septal defect or of a patent ductus arteriosus accompanying an atresia or a stenosis at the pulmonary orifice. The attacks may happen from 10 to 100 times daily. The survival of a child afflicted in this way is most improbable. Together with these signs, there is usually an abnormal percussion dullness to be found over the first and second interspaces to the left and above the heart's dullness when the **ductus arteriosus** has remained patent.

Physical signs which occur in patients suffering with congenital lesion of the heart are often most misleading because of their variability. Murmurs may be heard in systole or in diastole or in both; thrills may accompany these murmurs or they may be entirely absent. Localization of the murmurs and of the thrills varies, but it may give some indication of the nature of the underlying lesion; however, there is great uncertainty because of the many combinations in which congenital defects may occur. A thrill with a systolic murmur which is heard over the upper part of the precardium and which diminishes in intensity or becomes lost toward the apex of the heart is a phenomenon which should suggest the possibility of a **pulmonary stenosis** with an accompanying **septal defect**. The character of the septal defect, however, may modify the sounds so that the murmur will be best heard at other parts of the precordium. The usual site of maximum intensity for the murmur is high up over the second left interspace, sometimes even as high as the left clavicle. A systolic murmur of the same distribution may result from a patent ductus arteriosus. If septal defects are present alone, the murmur produced is usually localized lower down over the third and fourth interspaces, but when these lesions are combined with a pulmonary stenosis or an open ductus arteriosus, the distribution of the bruits is naturally wider, and the murmur is then heard not only over the first and second left interspaces but also over the third and fourth as well. The typical murmur of the open ductus arteriosus proceeds throughout the cardiac cycle. It has been likened to the roaring sound of a railroad train passing through a tunnel. When this roaring, to and fro murmur is heard, and it is possible to note a marked accentuation of the second pulmonary sound,

the presence of the open ductus arteriosus is certain. The accentuation of the second pulmonary sound enables the observer to distinguish the murmur of the **open ductus arteriosus** from the murmur of **pulmonary stenosis;** in this latter condition the second sound is very weak or is absent. Systolic murmurs, also accompanied by a systolic thrill, best discerned over the second and third left interspaces, are heard when there is **communication between the aorta and the pulmonary vessels.** Such communications result because of a defective development of the primitive aortic septum. When they occur, the second sound of the heart heard at the pulmonary area is well accentuated. Presystolic and diastolic murmurs are sometimes to be heard alone or combined with systolic bruits when septal defects are present without any other anomaly. There may be a diastolic murmur when the infant is held in the erect position and a systolic when it is laid recumbent, or vice versa.

Radiographic examination is of distinct value as an aid to differential diagnosis. An increase in the area of the heart's opacity confirms the physical signs of ventricular hypertrophy and of auricular distention. Marked widening of the shadow at the base of the heart indicates a dilatation either of the pulmonary artery or of the aorta. The shadow cast by the dilated pulmonary artery is distinctive. It appears as a typical bulge to the left and just above the heart shadow. It corresponds to the dullness to be found on percussion in the presence of a persistent opening of the ductus arteriosus. When the vessel shadows are well increased to the right of the median line about the same height, the opacity usually proceeds from the shadow cast by a dilated aorta.

Narrowing and deformities of the great vessels show as diminished or distorted shadows on the x-ray plate. Alterations in the shape of the shadows of the ventricles are characteristic of valvular lesions of postnatal origin; although the same sort of altered shadow occurs in the radiograms of pulmonary stenosis, of patent ductus arteriosus and of narrowing of the aorta. Septal defects are unlikely to cause an altered shadow form. When there is clinical evidence of these defects and an alteration in the radiographic shadow, it is usually because other anomalies are present and these complicate the shadows and obscure the readings. Under the fluoroscope, the disappearance of the normal auricular shadow with its characteristic fibrillary movement, and the appearance in its place of an equally characteristic pulsating

shadow of the right ventricle at the right border of the cardiac opacity, are evidences of a hypertrophy of the right heart and are of some value as an aid in the differential diagnosis of different congenital defects.

The *management* of infants with congenital lesion of the heart must of necessity be directed to maintenance of a hygienic environment, the assurance of the highest possible degree of resistance and the alleviation of the symptoms of inadequate circulation that may develop in those cases in which decompensation threatens or is present. In the earlier months of life, our effort is to guard the child from undue circulatory stress, whether this arises from crying, from respiratory disease with its attendant strain on the right heart, or from digestive disorders with flatulence and and encroachment of the distended stomach and intestine on the thoracic cavity. The weakness of circulation in the earlier months of life leads to a state of affairs which readily induces malnutrition and atrophy, and as passive congestion engorges the abdominal viscera as well as the lung, the processes of digestion may be feeble and uncertain.

All pains must be taken to prevent *pulmonary complications*, as most infants who succumb to congenital cardiac lesion die because an added burden is thrown on the circulation by pulmonary congestion in pneumonia or bronchitis. These are pathologic conditions to which the child is especially subject because of the abnormal circulation in the lungs which usually complicates congenital heart disease. Extreme care must be taken to prevent cardiopathic children from coming into contact with children who are possible carriers of the exanthemata and other infections. The provision of warm but not overhot living quarters is a necessity for children of this kind.

For an infant, it is important that the *nutrition* be carefully considered. It is wise to adapt the nursings of such children to their individual needs. Among the children who are fed with the bottle, it is the general experience that very dilute formulas are best avoided. The distention of the belly by overlarge meals or because of the production of flatus may cause the infant's death through embarrassment of the already enfeebled circulation. Flatulence in itself may be the result of a disordered circulation. This is especially true not only of children with congenital heart disease but also of other children with a congenitally inefficient heart muscle, a condition so often to be found in marantic babies. Infants who show pro-

nounced symptoms of congenital heart lesion should be fed rather concentrated formulas in small amounts. The food for such babies may well consist of partially skimmed milk with enough sugar or cereal added to bring the carbohydrate content of the food up to 7 per cent or 8 per cent. This mixture should be peptonized for at least twenty minutes. The food should be given in small quantity at a meal, not more than one-half of the volume that would be given a healthy baby of the same weight and age. In this way, as much food will be taken as would be given to a well child, but there will be only one-half as much water in the ration. It may be necessary in the early months to give even smaller feedings than this, and when such necessity arises, the child should be fed at a shorter interval than normally—perhaps even every two hours. The amount of food at a meal and the intervals between meals is not a matter of any great importance provided the child's 24-hour intake of food is sufficient to meet its nutritional demands.

As the child grows older, the time of peptonization may be diminished little by little until by the sixth month the milk is being taken without any predigestion. At this age, the milk should be boiled and cereal feedings carefully begun. During the second 6 months of life, egg yolk may be added to the diet. Fruit juice should be given early, and fruit pulp (prune or apple) be included in the meals at the usual time. When the simple fruit pulps are given, there is no need to give vegetable pulps. Sometimes children of inefficient circulation digest vegetables with difficulty. A trial of these may be made, and if the results are not satisfactory, vegetable feeding should be delayed until well into the child's second year.

The presence of *constipation* seems to have a distinctly deleterious effect on cardiopaths, and it should be combated in every way possible. Sunlight and fresh air are most important hygienic aids in the treatment of these children. Heliotherapy, if properly applied, is of value, although it is imperative to remember that infants of poor circulation are susceptible to variations in temperature and that both heat and cold are borne badly. Cold especially is to be avoided. The infant should be kept warm and dry.

To improve the *circulation* and to develop the nutrition and resistance of the child, methods of massage and passive movement are begun early. The massage should be gentle, and the passive movements few in number. These measures should be used more and more extensively as the child grows older.

For the treatment of acute paroxysmal attacks of *cyanosis* and *dyspnea,* which sometimes appear in congenital cardiac disease, no measure is of such value as the mustard pack. (See Methods.) The pack is superior to the bath because it can be given with less fatigue to the patient. Two small packs can be arranged on a table, one to contain the mustard sheet, the other a plain wet hot sheet. The child can be slipped from the mustard sheet pack into the hot sheet pack with very little effort on the part of the attendant, and very little fatigue to the patient. Beside the mustard pack, if the child is syncopic, strychnine may be used. This condition is one of the few in which strychnine is of real value in the treatment of infants. One-six-hundreth ($\frac{1}{600}$) of a grain stimulates the sensorium and reinforces the effect of the mustard pack. When abdominal distention is great enough to interfere with the diaphragm and to embarrass the respiration and circulation by mechanical pressure, injections into the triceps muscle of 2 or 3 minims of pituitrin may produce a prompt evacuation of the flatus and a flattening of the abdomen.

The future of an infant suffering from congenital heart disease is always a matter of much interest to the parents and to the attending physician. In certain instances of the disease in which cyanosis and other symptoms are present from birth, the prognosis is bad. In that group of cases in which the physical signs are dominant, and marked symptoms of cardiac weakness absent, the prognosis is good. Infants born with minor defects in the septa, with narrowing in the aorta and with patent ductus arteriosus without other defect usually attain adult life; often the cardiac lesion has little influence on their well being, although it may definitely interfere with growth.

When there is an extensive defect of the septum, such as is encountered in biloculate or triloculate heart, there is little expectation of life beyond the earlier months. Pulmonary vessel atresia also is one of the conditions in which expectation of life is limited. Infants born with these lesions rarely live beyond the end of the second year. However, even if there is narrowing, if there is a reasonable patency of the pulmonary vessel, adult life may be attained, but only on the condition that during infancy the patient is protected from respiratory tract infections which are especially fatal under these circumstances.

Transposition of the great vessels with the presence of intact cardiac septa, atresia of the tricuspid and of the **aortic valvular orifices** allow no hope for survival. All patients suffering from these anomalies succumb after a short term of life.

CHAPTER XV

DISEASES OF THE BLOOD AND LYMPHATIC SYSTEM

An examination of the *blood of the newborn* reveals a greater number of all the cellular elements and more hemoglobin than is found in the blood at later periods of life. Red blood cell counts range from 6,500,000 to 7,000,000 per cubic millimeter, white blood cells vary between 20,000 and 30,000 to the cubic millimeter and hemoglobin readings are often as high as 120. In these earlier days of life, the polymorphonuclear leucocytes are proportionately high, forming between 70 per cent and 75 per cent of the total white cells, and in the smears there will be found some nucleated erythrocytes.

The *normal* number of red blood cells is usually approximated within the first ten days of life. The proportion of the white blood cells diminishes slowly and reaches about 15,000 within the first month of life, at which level the count is maintained until later infancy; so that the discovery of a white blood cell count exceeding 10,000 per cubic millimeter is not an evidence of a suppurative process at this time of life. This high count is largely an index of the activity of the lymphopoietic tissues, for the number of lymphocytes per cubic millimeter rises rapidly to form from 50 per cent to 60 per cent of all the white cells in the blood, as contrasted with a 30 per cent content in adult life. The actual number of polymorphonuclear cells to the cubic millimeter in childhood and in adult life runs about the same, while there is added a large increase of lymphocytes peculiar to the blood of infancy. As a result, a diminution in mononuclear cells and an increase in the polymorphonuclears are significant of an infection in infancy; even more so than the mere increase in the total number of white blood cells would be.

Hemoglobin rapidly diminishes from the initial 120 per cent and by the end of the second or third month, it has reached the normal infantile level of 65 per cent to 75 per cent. The activities of blood destruction and regeneration result in the constant production of many young cells so that the presence of large and small cells, (anisocytosis), of cells irregular in shape, (poikilocytosis), and of cells that stain irregularly, (polychromatophilia),

is found in conjunction with erythrocytes of normal size which carry a deeply stained nucleus, (normoblasts). In contradistinction to normoblasts, the occurrence of large, irregular red blood cells possessed of deeply staining nuclei, (megaloblasts), is an index of abnormality.

Red blood cells are more vulnerable to pathologic influences during infancy than in later life and they regenerate much more rapidly after damage than do red blood cells of more mature ages. This fact may account for the brilliant clinical results that often follow the use of the cacodylate of iron in the malnutritions of babies.

The pathologic changes in the blood itself may be made evident in relation to the cells and the hemoglobin on the one hand, or to the fluid part of the blood on the other; this latter may be altered quantitatively or qualitatively. Quantitative changes largely depend on the water exchange and result in alterations of blood volume. Increase in the proportion of water without increase in protein concentration is probably a basic factor in the production of the infantile edemas. Those conditions in which the water content of the blood and its proteins are diminished with resulting low blood volume and a diminution in albuminous elements, are given a predominant place in the pathology of malnutrition and athrepsia, particularly by Marriott. The same investigator lays much stress upon blood concentration, loss of water and relative increase in protein content of the blood as an essential factor in the production of the alarming symptoms seen after diarrhea and after vomiting.

Increase in hemoglobin occurs rarely apart from polycythemia in which the red blood cells may be increased up to 9,000,000 or 10,000,000. This condition is found especially among infants suffering from congenital pulmonary stenosis and in those cases in which there is rapid loss of fluid from the body. Decrease in hemoglobin must reach a figure as low as 30 on the Sahli or Palmer scale before it becomes alarming; for the milder degrees of anemia respond well to injections of iron and arsenic. A decrease of red blood cells, especially if the count goes as low as 2,000,000 or 2,500,000, is of bad prognostic omen.

Diminution of white blood cells (leucopenia) is unusual during the first two years of life; even in typhoid fever, malaria and influenza, striking degrees of leucopenia may be absent. A count of 8,000 to 10,000 white blood cells in an infant of from 6 to 24 months is compatible with the presence of these infections. On

the other hand, huge increases in the proportion of polymorphonuclear cells occur with suppurative and infective disorders and an increase in the number of lymphocytes may be found in the early stages of pertussis, and throughout the less fulminating cases of tuberculosis and syphilis. However, in estimating a lymphocyte increase, the normally high lymphocyte count of the infant's blood must not be overlooked.

In considering the leucocyte count it should always be borne in mind that the estimate of the number of white blood cells may vary under different conditions other than disease processes. In the first place, estimation is subject to variations of technic; furthermore, the vasomotor control of the peripheral capillaries must be considered. It has been demonstrated that so great a difference as 5000 may exist in the count of the venous and capillary blood in the same infant; so that in the instance of a mild leucocytosis or leucopenia, too much importance should not be attached to the white cell estimation, especially if the differential count is within normal limits.

Newborn babies may bleed copiously from the mouth, nose or umbilicus, or into the skin. Voluminous hematemesis or the passage of large hemorrhagic stools may occur. These symptoms, singly or together, mark the condition known as *hemorrhagic disease of the newborn*. Untreated, the persistent bleeding leads to exsanguination and death. (See chapter on Hemorrhage.)

Repair of the blood of a normal infant after hemorrhage is rapid and complete as to restoration of cellular elements, slower in the increase of hemoglobin. After very severe hemorrhages, the generative powers of the hematogenic tissues may be so exhausted, the anemia so profound and the interference with body functions so extreme that death will follow. This happens in certain cases of hemorrhagic disease of the newborn that have not received prompt treatment.

The most important steps in the *treatment* of hemorrhage are to arrest the hemorrhage and to maintain the blood volume. Staying the hemorrhage in all cases where sepsis is not an etiologic factor, and even in some of these, is a simple matter easily accomplished by the injection of whole blood intramuscularly. By this process it is not necessary that the blood be grouped, donor with recipient, as is imperative in blood transfusion. Twenty to 50 c.c. of whole blood withdrawn from the vein of any one

not luetic, injected with a ground glass syringe into the triceps muscles, or in a child past the diaper soiling age, into the gluteal region, is a potent therapeutic weapon.

Blood transfusion as a means of combating hemorrhages and their results is a very effective remedy and it is not so difficult or complicated a procedure as is generally thought. In infants, the injection of citrated blood fills all the requirements of a blood transfusion. After animal experimentation, Sipperstein has adopted and recommends the transfusion of citrated blood into the peritoneal cavity. Brown of Toronto, after extended use of the methods, finds it can be used without harm to the patient. If experience continues to favor the method it will be a great advance because intraperitoneal injection is simply performed.

The two syringe method in practiced hands is a valuable and safe procedure which does away with the need of citrating blood. When dealing with an infant so young that the anterior fontanelle has not closed, this method is sometimes of distinct advantage.

Of the **secondary anemias** following infection, the most classical example is afforded by diphtheria. In this disease, there is no doubt that the toxin has a direct destructive action on the red blood cells and on the hemoglobin, for almost from the beginning, the erythrocytes and the hemoglobin begin to diminish, a process which then continues throughout the acute stage of the diphtheria and for some time following it. The same sort of toxic effect is demonstrated by the toxin of the pneumococcus, an organism so frequently and so viciously pathogenic for young babies.

With the exception of congenital syphilis, the secondary effects of chronic intoxications on the blood of infants are not great during the first year; bacterial invaders are either conquered or conquering with rapidity during this period. Toward the end of the first year and during the second, however, rachitic manifestations and the influence of low grade infections by the tubercle bacillus may be made evident by a persistent and increasing anemia of the secondary type. Congential syphilis, apart from the florid form may run a subacute or chronic course with little or no blood destruction and with but slight effect on the blood forming functions; so that the presence of syphilis is therefore compatible with a high degree of hemoglobin and well-maintained cellular content in the blood.

Intestinal parasites are not common during the first two years

of life. With the exception of ankylostomum, worms are not direct causes of anemia. This worm abstracts blood from the body by way of the mucous membranes that it wounds and at the same time produces an absorbable toxin. The combination of hemorrhage and toxemia is potent in producing blood destruction and the resulting anemia may be profound.

During the latter months of pregnancy, the mother supplies to the fetus an excess of iron beyond its tissue needs. This excess is stored in the liver and is utilized by the infant in its early months of life; apparently this is a provision of Nature to compensate for the fact that the natural food of the infant, breast milk, is deficient in this particular element. It is well known that premature babies are prone to develop severe anemias during the second half of the first year. It is probable in these cases that the maternal supply was insufficient for the postnatal purposes of the infant. This insufficiency of supply may be due to one of two things: either the child was born before iron storage was sufficient or else there was some interference in the normal process of storage. Once the inherited iron store is used up, the ingested foods must be depended upon to maintain the needed hemoglobin balance in the body; and those children who are not given a varied diet containing organic iron early in their lives, are apt to suffer from a lack of hemoglobin together with a diminution of red blood cells. For this reason, iron-carrying foods, such as egg yolk, spinach, apple, muscle and liver, should be given early. Properly prepared and properly presented, these foods will rarely disturb the digestion of any infant. The use of egg must be begun with caution for on rare occasions, an infant sensitive to egg protein may be encountered. However, if the method of hard-boiling the egg and creaming the yolk be used, practically all the egg white will be excluded; this is the part that sensitizes most children who show an anaphylactic reaction to eggs. Abt uses a nut-butter attachment on a meat chopper to reduce to a fine pulp the vegetables that he gives to babies. The result is a preparation so minutely divided that it can be passed through the nipple of an ordinary nursing bottle and can be given with the feedings of artificially fed infants. It is probable that secondary anemias can be prevented and if they are already developed, that they can be checked and often cured by the early use of mixed feedings in which iron and vitamine-containing foods are given due place. The canned spinach

and artichokes of standard brands are always obtainable and experimental work done in Whipple's laboratory at the Hooper Foundation indicates that the iron they contain is as available as it is in the fresh vegetable; even if their vitamines are altered, which we doubt, other sources of water-soluble vitamines, such as orange juice, tomato juice or potato puree may be used, and the iron these canned green vegetables contain, utilized.

The advantages of the administration of iron in medicinal form is a fact attested by many generations of shrewd clinicians. Although some men with experimental evidence in mind, stamp the procedure as unwarrantable, none the less, the conservative clinician does not abandon its use. The saccharated carbonate of iron is a preparation efficient, pleasant, and easily administered, and it may be given in doses of 1 to 3 grains according to the weight of the child. Its slight constipating effect may be neutralized by the addition of $\frac{1}{6}$ to $\frac{1}{2}$ grain of mercury and chalk powder. These powders may be given 3 to 6 times a day with great advantage to infants who are suffering from mild anemia.

When the anemia is part of a malnutrition and the child shows lassitude and lack of appetite and loss of tissue turgor, the addition of arsenic in small doses is often followed by a very striking and rapid improvement in the symptoms. The simplest and most effective way of administering iron and arsenic is through the injection of the cacodylate of iron into the triceps muscle, beginning with 3 minims of the 5 per cent solution for a 10-pound baby and 7 minims for a 15-pound child. The injection may be given daily for 5 or 6 days and then on every second day for a period of 10 days more. A second course may be given after a fortnight if necessary.

Pernicious anemia and **chlorosis** occur so rarely in infancy that they are negligible.

Aside from the polymorphonuclear leucocytosis in sepsis, the lymphocytosis in pertussis, the eosinophilia in intestinal parasites and the possible leucopenia in malaria, typhoid fever and influenza, the number and form of the white blood cells suffer few variations during infancy. **Leukemia,** a fairly common disease during childhood and adult life, is rarely seen during infancy. Comparatively few authentic cases have been reported. When the disease does occur, the diagnosis must rest on the blood picture and in the **myelogenous** variety in particular, on the splenic enlargement as well. In the **lymphatic** type there is seldom seen the glandular

enlargement that is so striking a feature in adult cases. The child usually shows a marked pallor. Quite often there are petechial or ecchymotic spots over the body that suggest hemorrhagic disease. The spleen is enlarged in both varieties, although it shows greater enlargement in myelogenous leukemia. The blood picture is characterized by a marked loss in red blood cells and hemoglobin and a huge increase in leucocytes which often run as high as 200,000 per cubic millimeter. In the myelogenous form of leukemia, much of the increase is due to the presence of large granular cells, myelocytes, although the polymorphonuclear leucocytes and lymphocytes show enormous increases. In lymphatic leukemia, the white cell increase is hardly so great, and the differentiation is readily made when it is seen that most of the white cells, at times as many as 90 per cent, are lymphocytes. An **atypical form of leukemia** in which there is a normal white cell count or even a leucopenia sometimes occurs. At first sight, these findings may be confusing but when it is observed that myelocytes are present or that there is a great increase in the proportion of lymphocytes found in the blood smears and enlargement of spleen, liver or lymph glands is noted, the diagnosis can be made.

Treatment of a case of leukemia is of little avail. Death usually occurs within a few weeks. Splenectomy, sometimes advocated in adults, is impossible in infants. The x-ray is said to give temporary relief by diminishing the size of the spleen and by reducing the number of myelocytes. Iron and arsenic have been used empirically with little advantage. The use of vaccines made from the streptococcus does not seem rational as it has not been established that this organism, sometimes found in blood cultures from leukemic patients is anything other than a secondary invader. The administration of benzol, a recent therapeutic venture in the treatment of this disorder, undoubtedly does reduce the number of the white blood cells, but this leucolytic action is of no avail as the disease is one of the hematogenetic tissues rather than of the white blood cells themselves.

Another disorder of the blood and hemopoietic system which it is important to differentiate is the "**splenic anemia**" of the English, the "**anæmia pseudoleukemica infantum,** (von Jaksch's disease)" of the Germans. The clinical picture of this disease is remarkably like that of a myelogenous leukemia. The differentiation lies chiefly in that it occurs mostly during the first 2

years of life and in that 60 per cent to 70 per cent of the cases recover. The symptoms consist of marked pallor, wasting and abdominal enlargement. The child is exceptionally languid, there is considerable edema, and petechial hemorrhages may be present just under the skin. The spleen is enormously enlarged and may seem to fill the entire abdomen. The liver and lymphatic glands are but moderately enlarged. The blood shows a marked decrease in hemoglobin even to as low as 20 per cent; a red cell reduction to 2,000,000 to 2,500,000 occurs, while there is a moderate leucocytosis, usually below 30,000, with the greatest proportionate increase in the lymphocytes; myelocytes are always found, although in much smaller proportion than in myelocytic leukemia.

It has been contended that this disease is an anemia in which the white blood cell count and the enlarged spleen are merely phases peculiar to the age of the patient. The fact that there has been a number of these cases reported in which there has been an absence of the common etiologic factors of anemia, speaks against this contention. The similarity between this disorder and myelocytic leukemia is to be noted, but the low myelocyte percentage as contrasted with a high percentage in the leukemia, together with the age incidence, is sufficient to insure differentiation. Diagnosis is further confirmed by the course of the disease; patients suffering from myelocytic leukemia invariably succumb while those afflicted with von Jaksch's disease most often recover.

The *treatment* consists essentially of close attention to diet and to good hygiene. Drugs are of little or no value and may be even harmful by aggravating the diarrhea which is often a troublesome complication.

Amongst the blood disorders, there are some that are not yet classified because of our limited knowledge in this field. Included among these is the condition called **purpura**, in which there is an extravasation of blood under the skin in small spots, (petechiæ), in linear markings, (vibices), or in diffuse areas, (ecchymoses).

In earlier medical times, many diseases in which the symptom of hemorrhage into the skin and mucous membranes was an outstanding feature, for example the severe exanthemata, were classified with the purpuras. As knowledge increased, these diseases were removed from this category. But there still remain a number of conditions in which the pathologic processes are not clear,

and to these the term, purpura, still applies. It is needless to say, therefore, that the etiology is as yet obscure. The streptococcus has been under suspicion for some time because of the fact that it has been isolated from the blood in some of these conditions.

Of the *secondary* purpuras, those of *infective origin* are commonest. They may occur in the course of measles, scarlet fever, or diphtheria. Typhus fever is always accompanied by hemorrhagic spots, and in epidemic cerebrospinal meningitis, the cutaneous hemorrhage is often quite profuse. It has been argued that the purpuras of rheumatic origin are produced by streptococcic emboli in the smaller vessels but this has never been demonstrated. The purpuras of cachectic, nervous, and chemical origin, interest the pediatrist but little. Pertussis may produce ecchymotic spots but whether this is due to toxemia or is produced by mechanical means or is a combination of both, is a moot question.

Cases of purpura in which the causative agent is not known are classified as **idiopathic**. In the simple purpura there are usually a few days of slight fever and indisposition and possibly some vague muscle pain is complained of by the older children, and then the mother notices the reddened ecchymotic areas. These clear up in a week or two and except in an occasional case that has recurrences, there is nothing more to attract attention. In the severer cases, the so-called **purpura hemorrhagica,** the child is quite ill for a number of days, there is vomiting and sometimes diarrhea and occasionally the passage of bloody stools. Hematuria and albuminuria are sometimes features. The bleeding is more extensive, and the course of the disease is longer than the simple form; sometimes it lasts several months, and shows a greater tendency to relapse. To the fulminant cases in which there is vomiting of blood and stomach contents and the passage of bloody stools, together with severe abdominal pains, the name **Henoch's purpura** has been given. This form of purpura may be complicated by intussusception.

In the idiopathic purpuras, the blood picture varies but little from the normal. There may be a slight leucocytosis, but even in the absence of other signs of a primary infection, it is of no significance. The only noteworthy blood change lies in a diminution of the number of platelets, but this finding occurs only in the severer cases.

The rôle that the hemorrhagic diseases play in cases of cerebral

hemorrhages in the newborn with the production of spastic diplegia is discussed in the chapter on Nervous Diseases. Suffice it here to say that the recent observations made by Warwick and Rodda, bring again into prominence the suggestion of Welsh made years ago that cerebral hemorrhage of the newborn may be in reality only a symptom of hemorrhagic disease. If this be so, the careful observation of newborn infants for the first few hours and their prompt treatment on indication of hemorrhages, will undoubtedly go far to prevent many cases of those distressing maladies, the spastic paralyses.

The *treatment* of the mild purpuras may be expressed in one word—hygiene. As there is no specific remedy, we must rely on proper feeding, fresh air and good nursing. The purpuric child should be closely watched and on the first indication that the case is to become more than a mild one, active treatment should be instituted. This consists of the injection into the triceps muscles or into the buttocks, of 20 to 30 c.c. of recently drawn whole human blood. Blood grouping is not necessary as the blood cells themselves probably play a minor rôle in causing the cessation of the hemorrhage. The only precaution is that the donor of the blood be not luetic. The mother's blood should be chosen if possible, the father's next of choice. For details of the technic, see chapter of Methods.

Hemophilia, a blood dyscrasia which usually makes itself apparent before the end of the second year of life, is characterized by uncontrollable hemorrhages. These are usually induced by wounds, even very slight ones, although spontaneous hemorrhages probably do occur.

The disease is thought to be hereditary in origin, because there are well authenticated histories of families of "bleeders." In these families, hemophiliac individuals appear in successive generations. It is a singular feature of the disorder that males, compared to females, are affected in a proportion of about twelve to one. In such families, the mother transmits the disease although she herself may be usually immune.

The disorder is first recognized by the occurrence of hemorrhages, seldom severe, but very persistent. The slightest scratch or abrasion may be the site of a continuous oozing for hours. Circumcision, a slight operation, may be the cause of intractable bleeding. Hemorrhages into the skin at sites in which it is certain there could have been no trauma may occur. Epistaxis,

bleeding gums, hemorrhages into the joints or viscera are often features.

The blood changes are not characteristic. There may be slight reductions in the number of the red blood cells and in the hemoglobin, due to the constant hemorrhages. The coagulation time has been said by some observers to be increased but this has been disputed by others whose authority carries equal weight. These variations of opinion may be due to the fact that there is no entirely satisfactory method of estimating coagulation time. Many theories have been put forward to account for the phenomenon of blood coagulation; of these, that which most satisfactorily illuminates the many isolated facts is the theory of Howell. This premises the factors of coagulation as 5 in number, namely: prothrombin, antithrombin, thromboplastin, fibrinogen and calcium. These are all present in the blood except thromboplastin which is found in the tissue juices. Within the blood vessels, prothrombin and antithrombin are held in combination so that there is no clotting. When blood is shed, the antithrombin is neutralized by the thromboplastin. The prothrombin is thus freed and becomes activated by the calcium. The thrombin thus resulting converts soluble fibrinogen into insoluble fibrin or clot.

This theory has formed the basis for investigations of hemophilia undertaken by Hurwitz. These have demonstrated that a marked deficiency of prothrombin is a constant finding in this disease, and Gelston has shown that a similar condition may be found in Idiopathic Hemorrhagic Disease of the New Born.

The prognosis in these cases is bad. While few die of the effect of hemorrhage itself, the general health is so interfered with that the child may easily fall a victim to some intercurrent disease.

The *treatment* during an attack of hemorrhage resolves itself into pressure on the affected part if that be accessible. This pressure should be just sufficient to produce an ischemia and it should be continued over a considerable period of time. If the bleeding part is not accessible, general measures must be resorted to. Of these, one of the best is the injection of whole blood as outlined above in the treatment of the purpuras. Gelatin has been recommended but it has the disadvantage that the product sometimes contains the living spores of anthrax. Maternal or paternal blood in sufficient dosage, 20 to 30 c.c. in an infant, is nearly always obtainable and it is easily administered;

the only equipment necessary is a sterile syringe and some tincture of iodine. Subcutaneous injections of whatever kind have the disadvantage of possibly starting fresh hemorrhages from the puncture wound, and this must be considered. Pituitary extract has been recommended for visceral hemorrhages as it has the effect of contracting the unstriped muscle fibers of the blood vessels. It is said to be contraindicated, however, in cases of bleeding from the kidney as it has the opposite effect on the renal vessels. In severe cases, the administration of carbon dioxide gas has been recommended as it produces transient increased coagulability. The use of thymus extract has been reported to have been of some value, and calcium lactate has its advocates. The general health of the patient should be built up by properly balanced diets, fresh air and careful hygiene. It is needless to say that in cases of families with a history of hemophilia, operative procedures should be avoided.

Lucas of the University of California has investigated blood conditions in the newborn, especially with relation to coagulation time and the elements concerned in its prolongation, or retardation. He has found that during the first few days of life, blood coagulation time is increased in normal children as well as in those with a tendency to bleed, and that this increased coagulation time seems to be due to a deficiency in prothrombin. (The insufficiency of the data in hand makes Lucas unwilling to accept this as the only factor in the changed coagulation time.) The problem is made more difficult by the presence of bile, in quite large quantities, in the circulating blood of many newborn. This apparent reduction in prothrombin is of great importance because of a similar finding in hemophilia, and in hemorrhagic disease of the newborn, except when sepsis is an etiological factor.

CHAPTER XVI

DISEASES OF THE NERVOUS SYSTEM

The nervous system of the newborn child is limited both in receptivity and in registration. Consciousness is present, but the special senses at best perceive imperfectly. Anatomically, the nervous system in a normal newborn child is complete in its elements; but certain processes, such as myelination of fibers with a consequent isolation of special paths, are incomplete. These paths act as conductors and coordinators of various neuronic activities, and in some parts of the nervous system their complete development is delayed for months. For this reason, **muscular incoordination** is normal in the newborn, and in many children, mild tremors and twitchings of the muscles occur; for this reason, too, evacuations of the bladder and bowel are matters of spinal control, and cerebral inhibitions, which later develop, are now largely in abeyance. Most forms of enuresis in later life are due to the persistence of the spinal type of bladder control.

It is characteristic of the newborn that the child reacts with great promptness by motor responses to sensory stimuli. Anything that touches the lips may produce the sucking reflex. Pressure or pain will evoke in the child a motor response into which all of the skeletal muscles may enter. But stimuli that can be recognized are few in number.

From birth certain aments are incapable of perceiving or reacting to irritations of the nervous system. In the normal infant, common sensations, especially those of hunger and thirst will be recognizable from the first days of life. The sense of taste, while far from exquisite, is undoubtedly present very soon after birth, although its acuity must vary with different infants, as some will object to ill-flavored substances that others will accept without protest.

After a few days of life, the normal baby should discriminate between the intensity of different sounds, and many little ones are frightened by banging doors and very loud noises. Light perception also is present early in life, but coordination of the ocular movements is a much later development and it is rare for a child

to recognize individuals and to follow attractive objects with his eyes until he is at least 4 months old, although occasionally this phenomenon may appear in children not more than 8 or 10 weeks of age.

Consciousness is a function of simple sensory registration. The emotions are modifications of consciousness which go hand in hand with increasing acuity of sensory perception and of a development of the apparatus which coordinates sensation with motor responses. During infancy and childhood, in the nature of things, the personality is much restricted. Janet teaches that hysteria in later childhood and adult life is the result of a reversion of the individual to this type of restricted infantile consciousness and that neurasthenia is due to the opposite state of affairs, that is, it follows on the development of expansiveness of the personality. In an infant, anger and resentment in response to personal discomfort are exhibited by typical motor evidences such as struggling, kicking or screaming.

In the earlier months of life, there is little evidence of pleasure. A well-fed, normal baby shows no psychical evidences of bodily well-being except sleep and contentment, but as it gets toward the end of its first half year, it does evince delight by cooing and smiling and by controlled movements such as waving its arms and legs and playing with its fingers and toes. Time is an essential element in the storage of sensations and their influence over the newly developed efferent tracts; so that little by little the personality of the individual is developed and with it certain mental faculties which seem to bear definite relation to the emotions.

The first of these qualities must be *memory*. The power of memory is shown by voluntary repetition of various movements which were primarily aroused as involuntary reflexes. Some children of limited mentality, repeat in a purposeless way, hour after hour, the same movement. Such infants fail to register the normal number of stimuli which are essential to the development of a full mentality. This limitation may be, and usually is, due to some definite physical defect in brain structure.

Next to memory, *will* is probably the earliest of the mental faculties to develop in infancy. The characteristic of will in infancy is persistency. Overpersistency can very readily develop into obstinacy. It is also typical of the infantile will that it is readily diverted by the wise presentation of an engaging interest,

a fact that will always be utilizable in the proper training of children. Attention and concentration are essential to the development of will and the infant is unable to exhibit will until he has developed certain powers of concentration. *Imagination* begins to develop towards the end of the first year and it is evidenced in the child's play. At first it is very simple, and most often its earliest evidence is seen when the child pretends to be taking food from an empty bottle, although it may be so surfeited that it may not desire to be fed. The development of personality continues throughout the period of individual human existence. The fundamental contributions, however, are made during the first 7 or 8 years and of these, those acquired during the first and second years are of greatest importance.

It is difficult for a medical man to accept any theory of normal nerve function or *functional disturbance* that is based upon the conception of inborn intellectual unit characteristics which are independent of the physical structure. Certain modern psychologists, affected by metaphysical considerations, are attempting to support a theory which classifies emotions and single intellectual qualities as inheritable unit characters, entirely independent of anatomical structure. On the other hand, most medically-trained psychologists believe that observation leads inevitably to the deduction that all nervous processes, including the mental, are the results of stimuli perceived and the translation of these through the motor apparatus into action; or when action is not induced, their storage as sensory impressions in memory, conscious or subconscious. The storage of sensory impressions and the ability to recall these and use them in initiating movements under the direction of the will, give a simple explanation, satisfactory for all normal and abnormal processes, whether they be purely nervous or mental.

Either the perceiving, the conducting or the registering apparatus may be *oversensitive or undersensitive*. Perception of stimuli may be excessive and the motor responses to them out of due proportion. Oversensibility may affect the whole tract or only parts of it. Increased perception of stimuli does not necessarily induce increased activity on the part of the motor apparatus but on the other hand, the normal perceiving apparatus may send nervous impulses to oversensitive motor neurons. In certain cases, the conductivity of the nerve paths between the receptors and the motor neurons may be in such a physical state that im-

pulses are carried imperfectly or the irritability of these paths may be such that small impulses that ordinarily are ineffective are carried forward in a manner sufficient to stimulate the motor cells and to produce muscular activity. Such a state of affairs is seen in spasmophilia and in tetany. Such hyperirritation is chiefly evidenced by cutaneous hyperesthesia, photophobia, intolerance of sounds, motor instability, in convulsions, and laryngismus during the age of infancy, as well as by chorea and tics in later childhood.

The influence of the *sympathetic* nervous system on the physical and mental functions is especially great during the first 2 years of life. It is the sympathetic that modifies the blood supply to the various parts of the body; undoubtedly the readiness with which the child flushes and pales, and the frequency of the minor symptoms of anaphylaxis so commonly seen in infancy, such as dermatographia, and cold hands and feet, are evidences of some slight abnormality in the sympathetic nervous control. In the more serious states, such as asthma and acute protein poisoning that occasionally appear to disturb the child, the rôle of the abnormally acting sympathetic is clear.

Increased irritability of the sympathetic is probably part of the explanation of the instability of the infantile emotions. It is characteristic of these emotions that their expression is explosive and excessive but without great persistency; it is the continuation of this type of emotional response past the first year that leads to the neuroses of older infants and children and even to those of the adults.

The proper *training* of the nervous system of children, especially of those who are hypersensitive, is an essential factor in the prophylaxis of neurosis. This training should be begun at an early age by means of simple measures, and it is important that it should be continued throughout the first 2 years of life, for it is in these years that the type of nervous reaction which is to become characteristic of the individual is very largely determined. It must never be forgotten that the training must be such that it arouses and fixes no antagonism on the part of the child, for many children are trained to be neurotics by overinterference with their normal nervous development. Because of human imperfection, every child will at times show habits that can be interpreted as neurotic, but most of these are automatically eliminated as time goes on, and further development

of the nervous and psychic systems takes place. For example, thumb sucking is a normal and not a particularly undesirable habit in young infants, but in most instances it is given up as the child develops a wider sensory field and the power to acquire other gratifications.

Habit is the result of repetition of certain sensory receptions followed by motor responses. It is well known that repetition renders efferent and afferent paths and receptors more sensitive and it is also clear that during the period while coordination of the sensory or motor functions is developing, we may expect the best results from attempts to guide the development of desirable habits.

The proper *environment* for an infant is one in which only a few sensory stimuli will reach it in the earlier months of life, although in the latter months, increasing numbers of stimuli must be permitted. The child should be so placed and its surroundings so ordered that these stimuli, so far as possible, may be kept under the control of the parents or attendants. This means, in practice, that the child should be kept clean and comfortable. The possible relationship between an irritable temperament in later life and physical discomfort throughout infancy is worthy of consideration. The intolerable weight of excessive bedclothing, not to mention the retention of body heat, may readily lay the foundations for an explosive irritability of temperament which coupled with improper training shows in later life in the disposition of the individual as a permanent defect.

The same result may follow from the practice of taking a child up whenever it cries, although in this case the physician certainly must sympathize with the mother of an irritable infant for it is most agonizing to the parent, to hear the continuous and poignant crying of an unhappy baby. Soon the crying becomes an index, not of discomfort, but of the child's will to be obeyed, and if the infant is fortunate in its mother, a struggle is initiated which leads finally to the knowledge that the mother is an authority to be respected; but too often, parents and grandparents, and nurses as well, give way and the child very soon comes to respect no authority but his own; just here may be laid the beginnings of an imperious self-willed temperament which has no toleration for discipline. This lack of respect for authority and intolerance for reasonable discipline is undoubt-

edly among the greatest of the social evils that plague us at the present day.

This and other similar indulgences are contributions to the development of self-centered, disagreeable personalities. One of the unhappiest examples of *bad training* among infants is the child who behaves as though the world were entirely centered about him, with an adoring family dancing attendance and giving way to every whim. If the attempt is made by an adult to assert his own desires, voluble and tearful protests or demonstrations of anger on the part of the child are followed by an abject surrender to it. In this group of circumstances lies one of the explanations of many neuroses that develop in later years, for in such a way, the developing mind is given so great a trend of self-will that it becomes overdeveloped in ego. Such an endowment insures those mental conflicts that are known to lead to the neuroses; for when an individual who has been trained in this way leaves the sheltered environment of which he is the hub, and participates in the stern realities of school life and later in those of the world, the conflicts may become so great that his nervous system is inadequate to perform its protective functions and becomes a menace rather than an aid to him. One of the most important services the doctor can render a family is by gentle friendliness, to protest this mistaken kindness, and to instruct parents of the dangers that lie along this path. Such parents are usually very conscientious and their error is caused through ignorance, and to show them their duty is often equivalent to accomplishing a cure.

To bring up a baby in a modern flat and do justice, especially if the child is the possessor of a full quota of proud relatives, makes a severe demand on the common sense and character of a young mother. On the other hand, a certain group of Spartan-souled young women, most of them college graduates, who, having studied all the wisdom of all the pediatricians, refuse anyone access to the baby, inevitably create a family situation that leads to great unhappiness and very often to irreparable breaches. The saving grace of common sense must be invoked in this as in all other instances. Most relatives are willing to forego the pleasure of handling and kissing the baby, if they are allowed to see it resting happily in its crib; if the child is kept as it should be, in a well-ventilated room or on a porch

apart from the usual living rooms of the house, it is a much easier matter to protect it from overattention.

Whenever possible, even in wintry weather, the baby should be outdoors several hours each day. The more delicate the baby, the more its need for fresh air. It is obvious that the infant must be kept warm. It is astonishing how sleepless, unhappy babies who have been kept in closed rooms or overprotected porches will blossom out when they are put directly out into the open air.

The modern perambulator in which so many babies are sent outdoors, is often the means of negativing the doctor's intention in the matter of fresh air. An examination of one of these contrivances will frequently show a hood almost impervious to air, drawn down behind the baby, and heavy curtains enclosing it in front in such a way that it would get more air in a ten by twelve room in a closed house. A simple box or basket on a stand with a parasol or small awning so placed as to protect the child from glare, is much better than any wheeled baby carriage, for which there is no need except the convenience of the mother or the nurse. There is advantage in doing away with the springed perambulator, for it removes the temptation to jiggle and jounce the infant, movements which tend to destroy tranquility and make the child dissatisfied with quiet.

It is hardly possible for a young infant to *sleep* too much in the early months of life; 16 to 18 hours sleep is not excessive, although many babies are content and do well with 12 or 14 hours. After the sixth month, 14 hours is enough, for now, waking hours are necessary for the development of intellectual capacity through the reception and the use or storage of sensory impressions. The hours of sleep, however, are as important for this development as are the waking hours, for unless proper rest is given, the nervous system becomes overresponsive to stimuli and in this way develops irreparable instability.

All social or household habits that infringe on the child's rightful hours of sleep are to be discouraged, although in some instances, their cessation may work an apparent hardship on the parents, especially on the father. Too often, sleeplessness and night terrors can be traced to the perfectly natural desire on the part of a father to romp with his baby after his return home in the evening. A reasonable association at a reasonable hour is to be encouraged, but excessive handling of the baby and romping and other boisterous entertainment are hurtful. While in one infant

such treatment may be tolerated, in another it may be the cause of night terrors, or wakefulness.

The presence of the infant's bed in the living room where the lights are lighted and the ordinary affairs of the household are passing, is another cause of bad sleeping habits. Through sheer weariness, the child may fall asleep, but such sleep is apt to be superficial and the habit of easy wakefulness is encouraged. Another common household habit, keeping the child in the parent's bedroom only to be awakened by the disturbance of their own retirement, may also be a cause of wakefulness at night. This is a problem difficult of solution for economic reasons, but it can usually be solved if the parents, interested in their baby's welfare, are reasonably careful. However, sometimes it will be necessary to provide a room adjacent to that of the parents' quarters in which the child can sleep undisturbed.

It is a striking fact that the child who does not have sufficient daytime sleep, sleeps badly at night. Many mothers attempt to improve the night's rest by keeping their babies awake during the day. This is a mistake; for the loss of day rest leads to overirritation of the nervous system with the result that the night sleep is neither so profound nor so restful as it should be. There is no better way of insuring quiet rest at night than to see that the child sleeps during the daytime in the open air. Many a child whose wakefulness has failed to respond to bromides or even to opiates, will sleep soundly when this simple measure is insisted upon.

A further encouragement to sleep will be found in the warm bath that may be given just before the child is put to bed. On the other hand, either a hot or a cold bath is stimulating and may keep the child awake. The sedative bath should be given at a temperature just below that of the body, say about 95°. For an infant, it should continue not over 5 or 6 minutes. Gentleness and a quiet environment during the progress of the bath will often help to insure a good night's sleep.

Free ventilation at night and proper bedclothing, neither too much nor too little, will also aid in securing restful and invigorating sleep. The all too common habit of loading an infant down with bed covers is to be discouraged. The old superstition that "cold" and "colds" are synonyms is still prevalent and no individual suffers from this misconception so much as the helpless infant

whose only means of protest is through restless kicking and crying for which he is accused of having the "colic."

The "cathartic habit" is a fertile source of wakefulness. Many attacks of intestinal spasm in infancy can be traced to insufficient doses of drugs especially of milk of magnesia. But it is undoubtedly true that when the child is constipated, if the stool has reached the rectum and lain there for some time without evacuation, it will produce distress that results in crying and loss of sleep. This condition can be easily obviated by the injection of a few drams of warm oil into the rectum, or the use of normal saline solution in the form of an enema. The prescription of a rational dietary will be essential to prevent a recurrence of the disturbing constipation.

Wool or silk-and-wool underwear is very distressing to some skins and undoubtedly plays a great part in producing uncomfortable, sleepless nights for the babies who wear this sort of clothing. The same statement may be made about the unnecessary, tightly-rolled binder of flannel which so many people consider an essential part of the infant's clothing. The rubber diaper protector is a modern refinement which can only be condemned; it produces discomfort because it is irritating to the skin of the buttocks, and also because it prevents normal heat radiation.

Soft mattresses stuffed with feathers or other such materials, down-pillows or oversized pillows of any sort, may contribute to loss of sleep by the facility with which the child becomes overheated. Conversely, insufficient bed clothing may lead to chilling, especially of the hands and feet, and may result in a similar disquieting wakefulness.

The presence of adenoids may account for restless sleep and in some instances be the cause of an intractable wakefulness. If the growth closes the pharyngeal end of the Eustachian tube, earache, due to increasing pressure inside the tympanum, may become a source of restlessness; the natural remedy is to remove the adenoid. As a palliative, a few drops of warm glycerin may be instilled into the external meatus.

Bedtime stories are not an advantageous form of entertainment for any child; but when children are old enough to listen, it is improbable that a mother, grandmother or nurse will entirely deny herself the pleasure of telling tales. Many childhood stories, originating in an age when child culture was little

understood, especially those with the highly tragic endings in which children so delight, must be interdicted whenever the physician is confronted by a case of insomnia or night terror. Oftentimes, a critical examination of the type of story told a child, whether at bedtime or during the day, will give a clue to the source of fright, which stored in the subconscious mind is released during sleep in the form of nightmare. The remedy is obviously the omission of such stories and their replacement by some more kindly type of tale of which there are many, specially devised for the infantile mind.

So obvious is the effect of diet, especially of the evening meal, on sleep and rest that little need be said, although in this respect, the nervous infant sometimes presents a problem of immeasurable difficulty. The size of the meal seems to have definite effect and with certain babies, an overlarge feeding is a fertile source of restless sleep; yet it is equally true that hunger may break the routine of the night and lead to wakefulness. Each child must be studied as an individual and be given the sort of evening meal best suited to its own requirements. Most often, however, as soon as the baby is deprived of its voluminous bottle feeding just before bedtime, and is given a more concentrated meal, disturbances of sleep disappear, and sleep becomes more refreshing. This is one more reason for the early abolition of bottle feedings.

The **examination of the nervous system** of all infants includes a search into the functions of the structures usually investigated when an adult is the patient but the inability of young infants and the unwillingness of those older to aid, renders it necessary to modify the procedures ordinarily used.

The first step is to have the child unclothed in a warm room. It is better that the clothes be removed before the physician's entry. Without touching the child, much can be learned, while the infant lies in bed or on the lap of its attendant. **Incapacities** or **peculiarities of movement** are readily noted, and **weakness, wasting** or **hypertrophy of muscles** may be easily seen. If the child is old enough to sit up, it should be encouraged to raise itself and the position of the umbilicus in relation to the costal cage should be noted. If the umbilicus moves upward during respiration, the lower abdominal muscles are at fault; if downward, the upper group is involved.

Placed on its back lying on a blanket on the floor, the attempts of the child who can stand and walk to get up and perform these

offices, will be instructive. **Motor instability, incapacity and incoordination** will be brought out; if **ataxias** or abnormal movements afflict the child, **tremors** and **athetoses** will be exaggerated and made clearly evident. Under such circumstances **hypertonia** with spasticity or **hypotonia** with flaccidity are readily noted.

Even in children who cannot stand or walk, valuable information about the condition of their musculature may be had by watching them crawl. **Cerebellar disease** especially may be suspected when the creeping child tends to fall to one side or the other. This test is a variation of Young's chair test, applicable to older infants in which the patient walking around a chair tends, as he turns its corners, either to tumble into or to fall away from it, the direction of the fall depending upon the site of the cerebellar lesion. **Spasticity** when it affects the hands or arms is readily noted by the position of the thumb which lies opposed to the palm while the fingers are more or less flexed, the forearm extended and the upper arm held tensely against the body.

The most notable postural sign of **spasticity in the legs** is their rigid extension and the adductor spasm which tends to bring them across one another, especially when attempts to walk are made. Overarching of the foot, hyperextension of the great toe and extension of the legs, usually accompany adductor spasm and rigid extension of the legs. Such a picture seen in infancy inevitably means damage or maldevelopment of the motor cortex or of the pyramidal tract; when it occurs without intellectual inferiority, this symptom-complex, is known as Little's disease. However, such a picture is often further complicated by the evidences of a feeble mind. Sometimes flaccidity of the neck muscles occurs because the cerebellum also has been involved in the process that damaged the cerebrum. **Choreiform twitchings** and **intention tremors** rare during infancy, give little diagnostic aid, but true **athetosis** offers fairly complete proof that the pyramidal tract is free from disease.

Before touching the child, indeed before attempting to have it stand or walk, observation should be made of the condition of the external **eye muscles** and of the **pupillary responses**. If power of lateral movements is lost, damage to the nuclei of the sixth nerve is diagnosable. On the other hand, should the vertical movements fail, disease about the third nuclei is certain. Weakness of one sixth nerve, if it is of sudden onset, may confirm the diagnosis of a meningitis if other signs of the disease are

to be found. But it must never be forgotten that strabismus, due to intrinsic muscular defect, is a common finding during infancy. Irregularity of the pupils and variability of the pupillary reaction are frequent concomitants of meningitis, especially of the tuberculous variety.

Weakness of the face, loss of power to suck well and strongly, deviation of the jaw to one side when an attempt is made to open the mouth, may occur. If these are observed, the inference is that the fifth nerve is damaged, and if anesthesia of the area supplied by the sensory part of the fifth nerve (cheek, nose, mouth) can be demonstrated, the diagnosis is certain. A careful observation of the movements of the circumoral and circumocular musculature while the child is laughing, crying and playing will reveal any weakness in these muscles which are supplied by the seventh nerve.

Hearing can be roughly determined by watching the child's response to the nurse's or parent's communications. During infancy, it is practically impossible to make any fine distinction or to draw diagnostic conclusions from the use of tuning forks, whistles and other instruments of the otologist. Neither is it possible to obtain valuable information from the vestibular tests. The same may be said of tests for taste and palate sensation which reveal evidences of impairment of the ninth nerve as well as of examinations for acuity of smell and for heminopsia, both so revealing when used in the case of older children.

After the observations just outlined have been made, elicitation of **reflexes** may be attempted. If the attempts are made part of a playful approach, the child will rarely resent them and accurate information may be had. The **triceps** and **supinator reflexes** are easily obtained in normal children. If on one or the other side, these are markedly greater, the inference is that a lesion of the cortex or of the pyramidal tract has occurred. Absence of these responses happens only in damage to the lower neurons and indicates poliomyelitis or some neuritic palsy such as may follow diphtheria. Brachial plexus injury may be the cause of a lost reflex.

Before the sixth month, the absence of **abdominal reflexes** has no significance, for many infants do not acquire them within their first half year of life. In children older than this, the absence of this reflex points to an interference with pyramidal tract conduction, although there are valid exceptions. Absence of

abdominal reflexes may be the result of a lower motor neuron damage such as may happen in a poliomyelitis when it attacks the thoracic region of the spinal cord.

Knee jerks are phenomena that are present in every normal infant from the day of birth. They are brought out with difficulty in certain infants, but most often, failure is the result of the imperfect methods used in attempting to elicit them. To be certain in its results, the method used must be one that obtains the greatest relaxation of the leg and thigh muscles. The best method of achieving this end is to have the child lying on its back and to support the thigh with the left hand while the leg rests on the examiner's forearm; the tendon of the quadriceps is then tapped by the fore or middle finger of the right hand or by a reflex hammer.

Marked increase in knee jerks, together with signs of spasticity, indicate involvement of the pyramidal tracts or of the motor cortex. Absence of the knee jerk is seen in poliomyelitis involving the vastus internus segment of the quadriceps, in diphtheritic neuritis, occasionally in a developing Friedreich's ataxia or in a myopathy. Knee jerks may be elicited with extreme difficulty or they may fail to respond at times even when the nervous system remains structurally intact. This is especially true in certain toxic conditions particularly in the pneumonias and in gastroenteritis. The presence or absence of the **Achilles jerk** has the same significance as the presence of a knee jerk except when the pathologic process is a myopathy. In such a case, one or the other of the Achilles reflexes may be absent. For the observation of this latter phenomenon, it is best to lay the baby on its face on the attendant's lap. Normally a slight tap on the tendo Achilles is followed by an extension of the foot.

Stimulation by stroking of the sole of the foot is followed in normal infants by a spreading out of the toes (fanning) and usually by an extension of the great toe in those who are unable as yet to walk. This is in contrast to the great toe flexion that answers like stimulation in older children and adults. Nevertheless, this rather momentary extensor response is so distinctly unlike the slow exaggerated, rigid, enduring response, **(Babinski's sign)** which indicates pyramidal tract damage, that the two are not readily to be confused. Therefore, Babinski's sign does provide valuable confirmatory evidence when one is searching for proof of pyramidal or motor cortex involvement in infants.

The feet should be warm when the test is attempted. The child's attention should be held by some one else than the examiner and it is important that a light stroke should be used on the outside of the sole passing from the heel toward the little toe.

The response to **Oppenheim's sign** is the same as that elicited by Babinski's technic, but it is produced in a different manner. The leg is held in a comfortable position and the thumb and forefinger is swept down the lateral margins of the tibia. When the fanning of the toes and extension of the great toe occurs, the sign is said to be positive. It is sometimes elicitable when the Babinski sign is absent although it has the same significance. **Chvostek's sign** consists of a spasmodic contraction of the face muscles after a sharp tap has been made on the side of the face just below the zygoma. It is found in tetany and in spasmophilia. **Trousseau's** sign may be observed in the same conditions. The reaction, a tonic flexor spasm of the forearm may occur when pressure is made on the bicipital groove. The reaction appears within 2 or 3 minutes.

Ankle clonus is a phenomenon that often can be demonstrated when the typical extensor response of the great toe is present. In attempting to elicit this reaction, the examiner lays the infant on its back, flexes the leg on the thigh and the thigh on the body. He then supports the leg by grasping it just above the ankle while he bends the foot sharply in flexion at the ankle joint. A slight but definite tension is maintained. If this measure is followed by rhythmical contractions, ankle clonus is present.

The presence or absence of the **cremasteric reflex** may be noted in male infants. It is best brought about by Purves Stewart's method in which the thigh is grasped and pressure made with the tip of the fingers on the nerve as it passes out of the femoral triangle. The reaction that follows is a prompt elevation of the testis of the same side.

The **abdominal reflex** is elicited by stroking the abdominal wall in each of its four quadrants while the child lies supine on an attendant's knees. Absence of this reflex is suggestive of profound cerebral lesions although local conditions, especially of the peritoneum, may prevent the response.

Brudzinski has added certain maneuvers of great utility for the examination of the nervous system of the child. One of these and the most important is the **Brudzinski neck sign.** It is a

method of determining hypertonicity of the extensors of the spine, hips, legs and head. When rigid extension of the neck is a feature of the clinical picture, attempts to raise the head by a hand placed under the occiput are followed always by a constant response; the head, neck and back move together as one rigid whole and as the trunk reaches a half-sitting posture, the thighs automatically flex at the hip joints and the legs on the thighs. Often too, there is an accompanying dilatation of the pupils.

The neck sign has the same significance as **Kernig's sign** in which attempts to extend the legs on the flexed thighs are met by resistance in the ham strings that renders any extension at the knee joints difficult, and full extension of the legs impossible.

These phenomena result from posterior root irritation. Increasing nervous irritability in these regions leads to hypertonicity of the extensors. They are always pathognomonic of meningeal irritation. Kernig's sign often is absent in the case of young infants with open fontanels and completely closed cranial sutures, but the neck sign is almost unfailing when meningitis of any origin is present. The **leg sign** of **Brudzinski** is also of similar significance. It is sometimes referred to as the **contralateral sign.** If, with both legs extended and the patient lying on the back, one leg is sharply flexed on the thigh and the thigh on the body, the opposite leg automatically follows suit.

The peculiarities of the developing uncompleted skull of the infant renders its careful examination a matter of importance. If palpation of the **open fontanel gives a tense sensation and the tissues over it are visibly bulging,** one may suspect an increase of intracranial pressure, although pressure may be much increased in young infants before bulging at the fontanel takes place. If such is the case, the palpating finger, run along suture lines will find abnormal separations there and at the site of the posterior and lateral fontanels as well. Percussion of the skull, under such circumstances, will elicit the "ripe watermelon" tone—an increase in dullness indicative of increased intracranial tension. Later in infancy, when the fontanels are closed a different percussory note may be heard, one of higher pitch **(Macewen's sign).**

After the child has learned to walk well, the gait may be of considerable significance. It may be **ataxic** or **spastic,** there may be unilateral or bilateral **palsies** or certain muscle groups may

be paralyzed; all these will direct the examiner's attention to various lesions of the brain or cord.

Changes in sensation are difficult to determine with any certainty in infants. **Anesthesias** and **hypoesthesias** are more apt to be discovered; especially the latter may be observed in profound degree in spina bifida of either the manifest or the occult type. **Hyperesthesias** and **hyperalgesias** are almost impossible to diagnose with certainty until the child has reached the age when it may cooperate with the examiner.

Eye-ground examination, especially in these days of the efficient electric ophthalmoscope, is one form of examination that should never be omitted during an appraisement of the infant's nervous system. In reality, it is easier to examine the fundus of an infant than of an older child. Hematropin is rarely necessary. The method of choice is that of Batten. The child is placed on its side on the attendant's lap. The head or lids are not to be touched. The eye to be examined is the upper one. Rarely will the infant close its eyes; if it does, it will open them again after a few moments. If the right eye is under examination, the observer uses his own right eye. The fundus seen and its condition noted, the child is turned over so as to bring the other eye uppermost. The left eye of the examiner is used to search the background and thus the examination is completed. The condition of the discs will confirm the other findings or it may reveal a state of affairs unsuspected before; atrophic neuritis or edemas of the disk, congenital defects or anomalies of the nerve or the vessels over it, choroidal changes, hemorrhages or engorgement of the veins, tubercles dotting the choroid, any of these may be observed. If there be opacities that render a view of the ocular chambers difficult, by resort to the indirect method of ophthalmoscopy it will be possible to show where the opacities lie, whether in the cornea, the lens, or the vitreous humor.

During infancy, **meningitis** and **encephalitis** and combinations of these are frequent. It is generally accepted that meningitis is but a phase of a generalized bacterial infection. In tuberculosis, meningitis is usually but a terminal event. Any pathologic organism may be causative but those commonly incriminated are the Diplococcus meningitidis of Weichselbaum, and the Bacillus tuberculosis of Koch; infrequently the Treponema pallidum produces a meningitis, and in young babies the pneumococcus is sometimes etiologic. Occasionally streptococcus, staphylo-

coccus, influenza bacillus, or the bacillus typhosus may be the organism that sets up the inflammatory process in the meninges.

The so-called **serous meningitis** and "**meningism**" are met with in infant patients; the latter especially during pneumonia and enteric disturbances, the former as a complication of suppurative ear disease.

Meningism is accompanied by all the symptoms of a meningitis but the character of the spinal fluid which flows through the puncturing needle under some degree of increased pressure, is found normal in its cell content and protein reactions. The condition is transitory and improves as the toxemia of the accompanying disease abates. In **serous meningitis,** the spinal fluid shows a high degree of pressure with some increase of protein content. Sometimes a very moderate increase in the cells is found but no bacteria are present, and there is no spontaneous web formation. Serous meningitis is probably never present apart from bacterial irritation. The bacteria may be circulating in the blood stream or they may be localized in a contiguous part as in an extradural abscess complicating an otitis. The symptoms are evidences of immunity processes by which the organisms may have been prevented from passing the meninges.

The diagnosis of meningism and of serous meningitis must rest on the clinical history and on the character of the fluid withdrawn by lumbar puncture.

Meningitis of meningococcal origin may occur as part of an epidemic or in more or less isolated instances, sporadically. Since we have acquired the conception that disease may be carried by persons, themselves immune, it is easier to understand the development of sporadic cases of meningococcus meningitis. This form of the disease is of especial interest to those who deal with sick babies, as three-fourths of the cases recorded as happening in childhood have occurred during the first year of life. Some writers attempt to separate the epidemic form from the sporadic type and attribute the latter to the ravages of a diplococcus not identical with the one etiologic for the epidemic form of the disease. This differentiation is based on cultural and fermentative peculiarities, peculiarities that have convinced the bacteriologist that there are many strains of this diplococcus, and impressed him with the need for a polyvalent serum in the therapeutics of the disease.

The affection reveals itself first by vomiting and by high fever, with urgent restlessness and sleeplessness. Often a rash is to

be seen. This may come in the form of hemorrhagic macules or there may be only a patchy erythema. The temperature curve is variable. In some cases, it ranges as high as 104° or 105° and remains constant until the signs of meningeal involvement appear. Once these signs come on, it is not unusual for the temperature curve to show marked oscillation, sometimes from a subnormal point to one as high as 105°. Often, however, even in the severest cases, the temperature does not exceed 102°.

Within 24 to 48 hours after the appearance of fever, convulsions are apt to appear. The younger the child and the higher the fever, the more probable is it that there will be convulsions. Between convulsions or replacing them when they fail to occur, there may be slight clonic contractions of arms and legs or of isolated muscle groups of the extremities or of the face. Early in the course of the affection, the fontanel will be found to be bulging. This sign rarely fails in infant patients and is one of the more helpful aids to diagnosis. Neck rigidity with a greater or less degree of head retraction is also a fairly constant early sign, although it may not develop for some days and in certain cases it may never appear.

The position taken by the child is usually on the side with legs and arms flexed. There is often great tenderness and the apparently unconscious infant resents any manipulation, especially if movement of the head results from it. Acuity of hearing and dullness of sight are curiously contrasting effects of the disease. Loud sounds seem to cause fright and distress while light perception is definitely lessened, so much so that observing the perfectly conscious patient one may gain the impression that he is blind, because while he lies with eyes open, he ignores any object brought into his visual range. This fact makes it easy to search the eye-grounds of such an infant. The pupillary reaction to light is however rarely lost. Ocular muscle palsy happens, but much less frequently than it does with meningitis of tuberculous origin. Choked-disk is seen in the later stages of this form of meningitis, but optic atrophy is rare. When it does form part of the clinical picture, permanent blindness may be expected to be a sequela of the disease. Blindness, like permanent deafness, is fortunately rare. Invasion of the eye by the meningococcus may result in opacities.

Respiration is accelerated, sometimes only slightly. In the earlier stages, breathing is not irregular, but later when pres-

sure on the bulb becomes excessive, as it does in cases which run a long course, sighing and phased breathing appear. In the same way, the pulse is rapid but regular, early in the disease; but later it becomes irregular and may become very slow in the chronic and in the subacute cases. Wasting is not a feature of the ordinary forms of meningitis because swallowing is rarely interfered with and recovery or death takes place before emaciation has time to become marked; but in those cases in which the meningitis has become chronic and in others where the inflammatory process has resulted in an obstructive hydrocephalus followed by opisthotonos, extreme wasting is the rule.

The patient's reflexes are all increased. Brudzinski's neck sign is almost always present and it is of value in arriving at the diagnosis. Absence of Kernig's sign is of little significance in infants with meningitis as it may or may not be present. The same statement holds for Brudzinski's contralateral sign. Oppenheim's and Babinski's toe signs are valuable aids when they are characteristic, but they may fail or they may be misleading.

The blood findings during the acute stages are those of a sepsis. A moderate to a high leucocytosis with a relatively high polymorphonuclear count is usually found.

The *spinal fluid* is pathognomonic in meningitis. Almost always, it is turbid. This turbidity may vary from a dull ground glass appearance to that of undoubted pus. It may even happen that the pus is so rich in protein that it clots spontaneously in the test tube. A cell count will show the presence of many pus cells (sometimes as many as 150,000 to the cubic millimeter). Of these, the greater number by far will be polymorphonuclear leucocytes. The protein tests of Nonne and Noguchi are positive and copper reduction is usually negative. If smears, made from the pus, fixed and stained by the Gram method, show extracellular and intracellular Gram-negative diplococci, there can be little doubt that the organism is the Diplococcus intracellularis, and that the patient is suffering from Meningococcus meningitis. When Gram stain is difficult to get or that available is of doubtful quality, it suffices to use methylene blue or Wright stain and if intracellular organisms are found, the same diagnosis may be made with a fair degree of assurance.

If satisfactory laboratory diagnosis can be made only after the lapse of some time, it must be remembered that in the presence of symptoms of acute meningitis, together with a spinal fluid that

is cloudy, we are warranted in making a provisional diagnosis of diplococcus meningitis and in proceeding at once to the injection of an antimeningococcic serum into the blood stream and into the spinal canal.

It must not be forgotten that cases of meningitis may be encountered before the organisms have passed from the blood to the meninges; so the disease cannot be excluded because one or two lumbar punctures have shown the spinal fluid to contain neither organisms nor any great increase in the number of cells. A later puncture may reveal pus and myriads of diplococci. In the same way, it is to be recalled that certain cases run on and become chronic. These may show a clear spinal fluid with only an occasional polymorphonuclear or nucleated cell, and they may reveal the organism only after a prolonged search through many well-stained films. Such cases may recover spontaneously or with the aid of appropriate serum treatment. Such a spinal fluid easily may be mistaken for the spinal fluid of tuberculous meningitis—a mistake which the long course of chronic meningococcus meningitis, together with the presence of symptoms of a chronic meningitis, tends to confirm. Probably not a few of the cases reported as instances of cured tuberculous meningitis were of this nature.

Herrick has drawn attention to the possibility that lumbar puncture may render the meninges more permeable and permit the invasion of bacteria circulating in the blood stream. While this danger has to be considered in cases of sepsis, the information derived by spinal puncture outweighs the disadvantages that may result therefrom and makes it possible and proper to do lumbar puncture when confronted with a suspected case of meningococcus infection in an infant.

Once diagnosis has been arrived at, no time should be lost in beginning *treatment*. Subcutaneous injections of 1 to 2 c.c. of serum should be given to desensitize the patient and a half-hour to one hour later 15 c.c. to 30 c.c. of fluid should be withdrawn from the spinal canal through lumbar puncture needle and slightly less than an equal amount of serum at a temperature of 100° allowed to flow by gravity through the same needle into the intradural space. (See Methods.) Serum which fails to agglutinate organisms cultured from the patient's spinal fluid will be ineffective. At the same sitting, or 6 hours later, a like amount of serum is to be injected into the blood stream by way of the longitudinal sinus, if the child is so young that the fontanel is

still open, or in the case of an older child, into a vein. When neither route is available, intramuscular injection may be used. Both injections should be repeated daily until the temperature of the patient becomes normal. As a rule, in favorable cases, this will take from 4 to 6 days; sometimes as many as 12 or 14 injections are needed. In the course of the treatment, symptoms abate and the fluid clears. If they are seen and diagnosed early and treated with reliable serums, at least 75 per cent of the patients should recover.

Fatal cases are of three types. First, those in which the patients succumb to an overwhelming toxemia within the first few days; second, a group that seems entirely refractory to the serum. These are probably infected with a diplococcus not represented in the strains used to prepare the serum that has been injected; and third, those that die after the development of a hydrocephalus. Of the cases that recover, a small percentage may show permanent blindness or deafness, some a mental defect or an epilepsy either alone or in combination; a few exhibit muscular wasting with impairment of gait.

Treatment, other than with specific antiserum contemplates maintenance of nutrition and of quiet for the nervous system. For the latter, a darkened room, a skillful nurse and if necessary an injection of $\frac{1}{16}$ grain of codein to a 20-pound child are useful. Vomiting, sometimes a very distressing complication, is best met by washing out the stomach with a 2 per cent sodium bicarbonate solution. If the vomiting is so severe that dehydration of the tissues follows, subcutaneous, intraperitoneal or intravenous injections of normal salt solution are indicated. Such injections are also of value in stimulating kidney activity and in reducing toxemia and fever. Gavage may be needed to maintain nutrition in those protracted cases in which swallowing has become difficult.

The symptoms characteristic of meningococcic meningitis are reproduced in instances of **meningitis due to pneumococci, streptococci, staphylococci** and the **influenza bacillus**. The differential diagnosis is to be made by the identification of the causative organism in the spinal fluid withdrawn from the patient. When pneumococci are found, a careful search sometimes will reveal a focus of pneumococcus pus in some site elsewhere than in the central nervous system; most often it will be in the pleural cavity, in the lung or in one of the epiphyses. In pneumococcic or staphylococcic meningitis, treatment is essentially symptomatic and it is invariably useless. Streptococcus septicemia may eventuate in a meningitis. The prognosis of the cases is not always fatal. It appar-

ently depends upon the virulence of the infecting organism. The records of the Children's Hospital in San Francisco show that one child who exhibited streptococci by blood culture and in the spinal fluid, recovered under symptomatic treatment together with daily lumbar punctures. One of the writers has had a similar experience with a patient in Lane Hospital and in another seen in private practice.

Meningitis due to the presence of the tubercle bacillus in the central nervous system presents a clinical picture frequently seen during the first two years of life, with its main incidence in the second year. The invasion of the meninges in this disease is but a final event in the course of a generalized tuberculosis. Therefore in contradistinction to the fulminant beginning of a septic meningitis, the onset of the malady is insidious and gradual in almost every case.

On taking the history of such a patient, it will usually be noted that there has been a period of some weeks during which malaise has been progressive. The hitherto happy child has become irritable; lassitude and drowsiness have been features, and emotional disturbances induced by trifling circumstances may have been noted. If the temperature has been taken, a moderate rise will have been found. Anorexia, an increasing photophobia with occasional vomiting, constitute the symptoms of the earlier stages of the disease. Even in this stage, an isolated convulsion may occur. After a few weeks of such a prodromal state, the signs of meningeal irritation appear. These may be introduced by a convulsion which may be the only attack of this kind to occur in the course of the disease. On the other hand, the occurrence of one or more convulsive seizures each day is commonplace, while there are cases in which the convulsions are so frequent that a status epilepticus is established. Very rarely, a case may run its course to exitus without a convulsion. Tremors and recurrent twitchings of muscle groups are characteristic of tuberculous meningitis. Facial muscles, a finger, the supinators of the forearm, a group in the legs, may at one time or other go into isolated contractions; first one and then another group may be affected and this variability is typical of the disease. Usually these muscular phenomena give way in the later days of the disease to a general muscular rigidity. Very striking is the involvement of the eye muscles. Strabismus, especially convergent, and involvements of the pupil with fixations, irregularity and inequality, are almost always found at one time or another in

well developed cases of tuberculous meningitis. These eye signs show the same sort of variability as do the irritable muscle groups, and the eye symptoms may vary from hour to hour.

The vasomotor system is involved and the vascular tone is affected so that flushings and pallors may succeed one another and the *tâche cerebrale* that results from stroking the skin under such circumstances is always to be elicited. Vomiting which is occasional in the earlier phases of the disease, now becomes a daily occurrence and together with it, develop other symptoms of intracranical pressure. Drowsiness, which may show a remarkable intermittence, headache and attacks of screaming in which the cry attains peculiar shrillness which has been denominated "cephalic" are marked features. The ophthalmoscope shows the effects of intracranial pressure in swollen nerve heads and distended vessels. Tubercles on the chorioid are stated by some authorities to occur in about 5 per cent of cases. Macewen's sign (a high-pitched note on percussion of the skull) is present in all cases of tuberculous meningitis in which sutures and fontanels are closed. In younger infants with these structures open, percussion gives a dull note that may be likened to the sound that is obtained when a ripe watermelon is tapped. In such infants, a finger held over the open, bulging fontanel, will appreciate a wave initiated by a finger of the other hand as it taps the head over the eminence of the parietal bone.

Throughout the disease, respiration is shallow but toward the end with increasing intracranial pressure, phased breathing takes place. It is not often a true Cheyne-Stokes respiration (in which the phase begins with a minimum respiratory excursion and increasing through a number of breaths to a maximum, falls off again to the minimum, halts a few seconds and begins again). In the phased breathing of ordinary tuberculous meningitis, the respiratory excursions are equal in length. They last a dozen breaths or so, then the breathing ceases for a few seconds and begins again—(Biot respiration). Changes in the pulse frequency and volume are also encountered.

The fever in tuberculous meningitis is ordinarily not high; 100° to 102° is the usual range but it is not unknown to find cases in which the fever rises to 104° or even to 105°.

The usual signs of meningitis are always to be found in a well-developed case. Brudzinski's neck sign, stiffness and retraction of the neck, abnormalities of the reflexes, sometimes increased, sometimes absent, contralateral signs, Oppenheim's, Babinski's,

all are to be encountered, although not always at the same time, in the disease. Like other signs of this disorder the reflexes show great variations from time to time.

In the face of the clinical signs outlined, lumbar puncture will establish the diagnosis with certainty. The fluid flows from the needle usually under pressure. Often it spurts with considerable force. Most often it is limpid, very rarely there may be some turbidity; often this turbidity is the result of contamination with a little blood. On standing (best in an incubator), a fine spider-web-like clot forms in nearly all cases. Such a clot forms in no other spinal fluid except that drawn from a patient with myeloencephalitis. Such a clot, carefully floated out onto a slide, allowed to dry spontaneously, fixed and stained by the Ziehl-Neelsen method should show the presence of acid-fast tubercle bacilli. The cell count varies from 50 to 500 cells per cubic millimeter, and the majority of these (70 to 80 per cent) are lymphocytes. Nonne and Noguchi globulin tests are positive and copper is reduced, especially if the fluid is drawn late in the disease.

It is rare that confusion in diagnosis occurs in a case of tuberculous meningitis. Once in a lifetime, a chronic meningococcic meningitis will present a puzzling problem but careful search will usually reveal intracellular diplococci. Encephalitis of the nonsuppurative type is more readily mistaken but the history of the attack with its sudden onset will usually suffice to differentiate an encephalitis. Care must be taken to elicit the history of the patient's past few months in detail, for it is astonishing how many times definite prodomal symptoms are overlooked by the parents and recognized only under the stimulus of questioning. Rarely a brain abscess will prove to be puzzling. The presence of ear disease or the history of its occurrence would aid in clearing the diagnosis. Brain tumors, without localization, are sometimes impossible to differentiate in the absence of a certain technic for isolating and identifying the tubercle bacillus.

There is no *treatment* for tuberculous meningitis. Our efforts must be directed towards obtaining as much comfort as possible for the infant. In the earlier stages, repeated lumbar punctures may be indicated in order to relieve the excruciating headache but once the child has become somnolent, relief of pressure can only aggravate matters by unduly prolonging life.

From the spinal fluid studies of Jeans and of Comby, one may conclude that more or less **meningeal inflammation** occurs as part of every case of **juvenile syphilis.** In its secondary stage, mani-

fest symptoms of meningeal involvement are, however, infrequent, but still they may occur often enough during the first year of life to warrant consideration for this form of meningitis.

The appearance of the symptoms is delayed until the third or fourth month of life. The earliest sign noted, may be a bulging of the fontanel, and a slight degree of hydrocephalus. Intellectual development is retarded and as a result the patient is apathetic and quiescent. Sometimes rigidity, simulating Little's disease is a feature and almost always recurrent convulsions occur. At the same time, the skin or eye manifestations of a florid lues may take place. Lumbar puncture reveals a clear fluid under some pressure with a cell content predominantly lymphocytic. This fluid reacts positively to the Wassermann test.

The treatment is that for syphilis. Mercury may be given by injection; arsphenamine injected into the vein is advantageous. Under the most intensive treatment, however, many such cases are refractory. Untreated patients may die or improve somewhat and later develop into juvenile paretics, or show some other form of cerebrospinal lues. Syphilitic infections share with meningococcic invasions the responsibility for many of the cases of acute hydrocephalus that develop during infancy.

Brain tumors, and intracranial abscesses are exceedingly infrequent during the first two years of life. Except for the more abrupt onset of an abscess, the signs of the two conditions are almost identical. Localizing signs are less evident at this period than in later life. Malaise, irritability, optic neuritis, persistent vomiting and the bulging fontanel of increased intracranial pressure are the revealing symptoms. A temperature line that remains at or below the normal may happen with either abscess or tumor, although the persistence of a subnormal registration speaks for abscess especially if suppurative disease of the ear exists or has been a preceding event. Abscess is wont to localize in the temporosphenoidal lobe or in the cerebellum. The later stages of both abscess and tumor are marked by increasing drowsiness, progressive slowing of the pulse, and final coma. Signs pointing to the involvement of the former site are unilateral changes in the reflexes, slow extensor response of the toe when the sole is stroked, increased response to a tap on the patellar tendon, and an absence of the homolateral abdominal reflex or at least its diminution.

When a one-sided ataxia is a feature and there is a nystagmus

and when the eyes are turned to the ataxic side, **cerebellar** localization may be suspected. Sudden death may supervene in the case of a cranial abscess, especially if it is localized in the cerebellum because of the facility with which the bulb is impinged upon. Death in such a case results from acute respiratory failure. When the intracranial abscess is small and becomes encapsulated, it is, in reality, an intracranial tumor.

Intracranial tumors are rare in any period of childhood and only about 10 per cent of those described have occurred before the end of the second year. At this age, the cardinal symptoms of intracranial pressure, headache, vomiting and optic neuritis very readily may be overlooked.

In young infants, headache is usually indicated by rolling the head or beating it on the pillows or by the carriage of the hands to the head or by the child's unwillingness to have the head moved, and by its resentment to bright lights and loud noises. In young infants, the disease may run its course without vomiting, and even when vomiting is present, the frequency of this phenomenon from other causes at this time of life, makes this symptom confusing instead of elucidating. However, if it be recurrent and without apparent relation to digestive disturbance, the possibility of the emesis being a part of the clinical picture induced by brain tumor should not be overlooked. In the presence of such vomiting, other evidence of brain tumor should be sought.

More than one-half of the tumors of the brain that have been found in infants, are tumors of the base, and of these, 45 per cent of the total have been in the cerebellum. This fact, together with the accessibility of the cerebellum, renders tumors in this position of special interest. Such tumors may be in the lateral or middle lobes of the cerebellum. They may be intracerebellar or extracerebellar. For the most part, they are of intracerebellar origin and are of the lateral lobes.

The characteristic symptom that permits the localization of the tumor in this region is the ataxia on the side of the lesion. The infant will show marked inability to reach out and grasp desired objects. Hypotonia on the same side is evident and characteristic. There is a tendency for the head to be turned in such a way that the ear is approximated to the shoulder and the face is turned to the opposite side. This position of the ear to the shoulder may or may not be on the same side as the tumor. If the child is old enough to walk or to creep, it will show a tendency to fall toward the side of the lesion. In creeping in-

fants, the buttocks tend to fall to the side of the lesions. Ocular palsies are commonly seen as the results of tumor in the cerebellum. Weakness of the external rectus on the side of the lesion, weakness of conjugate deviation to the same side or in the absence of this phenomenon, nystagmus, when the eyes are turned to the same side, are common manifestations. Cerebellar tumors are prone to produce early and extensive optic neuritis. As a rule, the reflexes remain normal until late in the disease when the knee jerk may become abolished. However, there is great variability in the patellar response. It may be found at one time, be absent a little time later and still later be found to have returned. There is no reasonable explanation known for this state of the reflexes.

When the **tumor** is outside the cerebellum, it is most often situated in the **cerebellar-pontine angle.** Beside the signs outlined as characteristic of cerebellar tumors, new growths at this site give evidence of interference with the seventh and eighth nerves whose exit is in the cerebellar-pontine angle. As a result, facial palsy and nerve deafness ensue. In infants it is difficult to be certain of the presence of the latter. Beside the seventh and eighth nerves, the fifth and sixth nerves may be affected by pressure, resulting in facial weakness on that side. In addition to these signs of cerebellar involvement, there will be weakness on the opposite side of the body with an increase of the deep reflexes, ankle clonus and Babinski's sign, indicating that pressure has involved the pyramidal tract as it passes through the pons.

When the **tumor mass** is situated in the **middle lobe of the cerebellum,** there are no specific localizing signs. In one case of the writers', the child tended to fall backward and the arm was elevated and the hand held above the head persistently, whether the child was supported in a standing position or lying on its back at rest, in bed. For the most part, the symptoms are in abeyance until the lateral cerebellar lobes or the pons have been pressed upon sufficiently to produce symptoms referable to these structures.

After the cerebellar, the next most frequent tumors of the brain are the **pontine tumors.** In the final stages of both cerebellar and pontine tumors, bulbar symptoms are put into evidence. They may result either from pressure on the bulb or from the invasion of the medulla by a continuation of the growth from its original site. The early signs of pontine involvement

are expressed in squint and crossed paralysis, that is, the face on one side and the arm and leg on the opposite side are affected. The facial paralysis is due to involvement of the lower neuron and is therefore flaccid, and as a result the child has difficulty in closing the eye. When the fifth nerve is involved, in both motor and sensory portions, the result is a weakness of the masseter muscles evidenced by the movement of the jaw toward the paralyzed side when an attempt is made to open the mouth. The sensory phenomena may be easily overlooked in an infant, but a careful observation will note quite early that the corneal reflex is absent and it is probable that injury of the cornea by foreign bodies may occur and lead to an ulceration.

As a stage in the progress of cerebellar or pontine tumors, *medullary symptoms* may appear or these may follow the presence of a small tumor originating in the bulb. Under the latter circumstances, death is apt to occur suddenly before many symptoms are noted. In the presence of a case of slowly growing tumor or of one lying outside the bulb, the striking features are the loss of the lateral movements of the eyes, facial paralyses, difficulty in swallowing due to paralysis of the constrictors of the pharynx and palate, voice changes due to paralysis of the vocal cords, weakness of the sternomastoid muscles and the upper portion of the trapezius muscle, and a wasting of the musculature of the tongue.

The development of a one-sided, external rectus strabismus in an infant with previously normal eye muscles who also has difficulty in closing the eye, and some weakness about the mouth on the opposite side, should lead the observer to suspect the onset of a pontine tumor. At a later stage, head retraction, rigidity with spastic paralysis of one or both lower limbs with or without alterations in the reflexes and a palsy in one or more cranial nerve distributions, may be added to the earlier signs and confirm the diagnosis of pontine tumor.

Tumors about the **basal ganglia**, the **optic thalamus** and the **corpora quadrigemina** first manifest their presence by alterations in oculomotor function; a ptosis, external strabismus, impaired power to move the eyes vertically, with a maintained ability to make lateral movements, is characteristic, and points to involvement of the nucleus or fibers of the third nerve. The pupils are dilated and they fail to react to light, sometimes to accommodation as well, the latter feature depending upon the

direction of greatest growth. Rhythmic tremor, muscular weakness, hemiplegia or diplegia with rigidity may complicate the picture if the pyramidal tract is involved. Like symptoms of motor-oculi disturbance, when these occur together with general muscular flaccidity and hypotonia, they suggest that the incidence of the new growth has fallen on the cerebellar structures.

There is no *treatment* that can be of the least avail for the relief of bulbar or pontine tumors. Our only hope, when these symptoms are met, is that the original cause of the trouble lies in the cerebellum where we know that surgery has some chance of reaching and removing the offending masses; and even when we have such knowledge, we must not be too sanguine, for many of the localizable cerebellar tumors are infiltrating gliomas, inaccessible to surgery. The operation of decompression, so valuable and so essential in cases of older children and in adults with brain tumor is rarely advisable during infancy because at this time, the readily separable sutures give way and the baby achieves its own decompression.

Infantile paralysis (poliomyelitis, polioencephalomyelitis) is a generalized infection in which, however, the nervous system suffers more specifically than any of the other tissues. It was first recognized as a poliomyelitis in which the inflammatory processes interfered with the anterior horn cells of the spinal cord and produced a flaccid palsy of certain muscle groups. These palsies are not uniform, some involve only a few muscles or muscle groups and others the entire musculature of a limb. The trunk muscles and facial muscles are also, in rare instances, involved, but for the most part, the arms and legs suffer because the lumbar and cervical enlargements are sites of predilection for the virus of this disease. The group of flaccid paralyses due to anterior horn-cell damage, still today provides the largest number of victims of the disease. But recent experience has shown that any part of the nervous system may be involved and that the nonsuppurative encephalitis, first described by Strümpell is the result of damage to the brain by the organism, as yet unidentified, which produces poliomyelitis. The work of Flexner, Noguchi, Levaditi and Landsteiner has given us reason to believe that the infecting agent is an ultramicroscopic virus with many of the characteristics we know inhere in virulent organisms. Like the virus of meningitis, this infecting agent reveals itself sporadically or in epidemics, and the incidence of its pathogenicity falls chiefly on

the young. By far the greatest number of acute manifestations of the disease occur before the third year of life, and there is reason to believe that older children who are themselves immune, may act as carriers of the infection.

The study of the disease is hindered by the character of the infective virus and by the fact that few experimental animals are susceptible to it. Primates alone are of value as experimental animals; the expense, and difficulty in obtaining them, limits the possibility of experimental work to a few highly endowed institutions. However, there are about 60 epidemics of the disease on record in different parts of the world, and the later ones of these have been exhaustively studied from clinical and epidemiological points of view. Sporadic cases are constantly under observation and practically every possibility of origin has been excluded except contact infection. Wickman in studying the Swedish epidemic in 1905 made one of the most fruitful clinical contributions when he recognized that besides the ordinary spinal form with its flaccid palsy and the encephalitic variety described by Strumpell, other clear cut clinical pictures follow from the infection when its main incidence falls upon various other portions of the nervous system. He, perhaps with an excessive zeal for classification, describes a spinal, anterior horn cell type; an ascending type similar to a Landry's palsy; a bulbar-pontine type; a cerebellar type in which acute ataxia is a feature; a neuritic type in which pain and tenderness is striking (and which in the last American epidemic was of frequent occurrence;) a meningitic type; a mixed type in which symptoms of two or more groups predominated; and finally, an abortive type.

The general symptoms of the disease are variable, depending upon the site of the involvement and upon its severity. In many instances, the parents have no knowledge of the sickness until it is discovered one day that the child is unable to use one or another limb; then on consideration, it is remembered that there was some slight malaise and irritation or perhaps a condition that might have been a fever. On the other hand, in the severe cases, the onset is sudden, lasts some days with drowsiness, great pain in the back and legs, and during this time, movement is resented.

There will be convulsions in some instances. This phenomenon is more frequent in the encephalitic and meningitic types of the disease. Vomiting and diarrhea are very often striking and distressing features of the onset of the severe cases in young chil-

dren. When the involvement is chiefly of the brain, there will be nuchal rigidity and retraction, a picture that is difficult to differentiate from that of acute meningitis. In infants, the loss of sphincter control is frequent but it is often overlooked because of the age of the child. Various rashes have been described; they always appear at the onset of the disease. There may be a scarlatiniform rash which, taken together with the evidences of extreme toxemia, make it impossible certainly to differentiate the condition from scarlet fever until time has elapsed for study of the symptoms.

Thomas Barlow has described certain cases of poliomyelitis, more frequent in the epidemic than in the sporadic form, in which the temperature persists and in which there is a tendency for the paralysis to extend suddenly after a few days quiescence. This alternation of events may go on for some time and on the whole, the cases resemble those that Wickman has classified as of the ascending type.

The deep reflexes of the affected parts are abolished and sometimes the superficial reflexes as well. The latter return with the general improvement rather rapidly but the deep reflexes remain absent for a long time and often they never recur. Paralysis of the flexor muscles with maintenance of power in the extensors may give rise to a pseudo-Babinski sign although in some cases involvement of the pyramidal tract may occur and a true Babinski phenomenon be found in the spinal form of poliomyelitis. The acute stage of the disease runs a variable course and may last for a few days or may persist for weeks. During this time, there will be little or no improvement and there may be retrogression. Sometimes there is a period in which improvement in the power of the paralyzed muscles begins and continues occasionally, to complete recovery. Most often, however, the recovery is at best but partial, although there is often improvement for as long as one year or even two years after the acute onset of the attack. It is during this stage that those muscles which show no tendency to recovery, begin to atrophy and at this time, vasomotor disturbances are made evident by blueness and coldness of the limb. There comes a time when atrophy on the one hand and improvement on the other, cease, and the full extent of the damage that the child has suffered, now, for the first time, is apparent.

The electrical reaction to the faradic current is lost while galvanic excitability remains persistent, at least until the later

stages of the paralysis; however, it may then completely disappear, but more frequently under galvanic stimulation, the affected muscles exhibit the reaction of degeneration. In the case of those muscles which are improving, the return of the muscular response to the faradic current is delayed long after a fair amount of voluntary power has been acquired. The presence of a faradic response in a muscle that has been paralyzed is of good prognostic significance.

When in the course of an infantile paralysis case, palsies of the cranial nerves appear, especially of the seventh, evidenced by weakness of the facial muscles, it must be concluded that the disease has attacked the brain in the region of the pons or of the medulla. During an epidemic, an isolated palsy of the third, fifth, sixth, and seventh nerves together with the symptoms of an acute illness, should suggest the possibility of the *bulbar form of poliomyelitis*. The symptoms of involvement of the third nerve are ptosis, loss of power of movement of the eye, dilatation of the pupils and loss of reaction to light and accommodation. Complete recovery is the rule. When the fifth nerve is involved, the weakness is shown by the masseter and temporal muscles which permit the deviation of the jaw to the affected side, when the mouth is opened. Difficulty in swallowing should be watched for, and when it is found, and especially if the child is old enough to talk, and the symptom is accompanied by speech defects, it may be accepted that the nuclei of one of the other cranial nerves, the ninth, tenth or eleventh have been invaded. The rapid and general involvement of the bulb is a terminal event in many cases of infantile paralysis of the progressive type. When the incidence of the virus of polioencephalomyelitis is chiefly on the brain, the disease is almost invariably ushered in by convulsion, loss of consciousness, and high fever. That part of the brain most extensively damaged is the motor cortex and if the child survives the acute stage, hemiplegia may result, and leaves behind a clinical picture indistinguishable from cerebral spastic paralyses of other origin.

Following a convulsion and loss of consciousness, there may be *aphasia*. Certain cases of *blindness* which have followed attacks similar to the acute manifestations seen as preliminary to these cases of hemiplegia, are attributed to an infective invasion of the occipital cortex of the brain by the virus of infantile paralysis. The same source of infection is also incriminated as the cause of

damage to the frontal region of the brain and the production of a *mental deficiency* coming on after an acute illness, accompanied by convulsions and high fever with loss of consciousness. After a similar attack, certain children are found to be **ataxic** without impaired intelligence, sometimes with a definite nystagmus. Such manifestations are attributed to cerebellar damage in the course of an acute polioencephalomyelitis and of such cases, a considerable proportion occur during the second year of life. If the child is old enough to have talked, the speech may become slow and deliberate or it may be entirely lost. Recovery in these cases cannot be prognosticated. Some of the patients recover in a short time, some after long periods, and some not at all. Certain of the patients in whom symptoms of brain damage appear, present clinical evidence of meningeal involvement. After a preliminary stage of drowsiness and irritation with more or less rigidity of the neck, the child may develop a convulsion and pass into a state of coma. Such a case may be readily confused with a case of tuberculous meningitis, and the spinal fluid withdrawn lends little aid to the differentiation because it has all the characteristics of the fluid found in tuberculous meningitis; that is, it is under increased pressure, shows an increased globulin content and a cell count in which, among the increased cells, lymphocytes are predominant. Furthermore, on standing, such a fluid often precipitates a fine spider-web-like clot identical with the web of a tuberculous meningitic fluid. The differentiation of the two fluids must depend on our ability to identify the tubercle bacillus when it is present in the spinal fluid, an identification that is not always easy to accomplish.

Most cases of **infantile paralysis with bulbar involvement** are rapidly progressive. Usually the medulla is attacked early and the patient dies in a few days or even after a few hours. On the other hand, a moderate proportion of those attacked recover and undoubtedly some of the patients who have been reported as having recovered from tuberculous meningitis, were in reality, sufferers from polioencephalomyelitis.

An interesting manifestation of infantile paralysis was described by Reginald Miller who showed that certain *acute tremors* of great rarity arise from an interference with the function of the cerebrorubrospinal system of fibers when these had been damaged by the virus of this disease.

In the presence of an epidemic, it is wise to recall that Wickman laid great stress upon the **abortive form of the disease.** In these

cases, there is reddening of the fauces with diarrhea, malaise, headache, fever, muscular pain and tenderness, and sometimes convulsions. Such an attack may end in recovery after two or three days' illness; but importance of these cases lies in the fact that possibly they may supply carriers of the disease.

For purposes of *treatment,* infantile paralysis should be regarded as an acute infectious disease and therefore the patient should be isolated. As it is known that the period of incubation is from 4 to 8 days, a 10-day period of isolation should be imposed on all contacts. How long the patient himself is infective is unknown but modern practice is to insist upon his isolation for at least 3 weeks after nasal discharges have disappeared.

During the acute stages of the infection, the essential feature of treatment is rest in bed. The room should be quiet, and it should be warm and well ventilated. It is important that all body discharges should be sterilized, nasal secretions especially. These should be received on paper napkins and at once be dropped into a paper bag, and the bag and its contents burned. In every case, no matter how slight the involvement, orthopedic experience insists on the necessity of *prompt fixation* of the affected parts. The advantage of the fixative apparatus is that it prevents undue stretching and minimizes subsequent deformity. When such apparatus is used, pain is not a marked feature. A light plaster cast is useful for this purpose. Small doses of aspirin or of acetanilid with caffein will often control the pain, but as the acute stage of the disease is of short duration, it is best not to temporize but to relieve the pain and secure rest by the use of opiates. Injections of codein or morphin ($\frac{1}{16}$ grain of the former or $\frac{1}{24}$ grain of the latter for a 25-pound child), are preferable. It is best to give a single ample dose early rather than to repeat small doses. The rest and immobility that are induced are valuable in the relief of restlessness and pain. Whether or not hexamethylenamin is of value is a moot question but as its use is advised by most authorities, it may be tried. The same attitude may well be taken toward the subdural injection of adrenalin as advocated by Meltzer. He recommended these injections after he had reviewed the work of Clark who used the drug intrathecally in cases of poliomyelitis on experimental animals.

The nutrition must be carefully supervised, especially in the case of those children who have difficulty in swallowing. It

may often be necessary to resort to gavage. It is well to recall that in all cases in which swallowing is impaired but still possible, it is of advantage to feed smooth, semisolid foods rather than solids or fluids, and if the child is placed with the head and shoulders low in the position chosen for feeding children who wear intubation tubes, swallowing is facilitated, because in this position gravity carries the food past the glottis and choking is prevented.

In the secondary stages of the disease, the *treatment* is directed to the paralyzed muscles themselves, and to the maintenance of the warmth of the limbs. Contractures and deformities are anticipated and prevented by the use of light splints or of plaster bandages. Massage, passive movements and electricity are used to stimulate and to keep tone in the muscles until the acute processes have subsided in the cord. The value of electricity is questioned by some authorities of great experience, among them Lovett of Boston, but its use in selected cases has seemed to the writers to be of value. Butler has pointed out that it is important to use that form of current to which the paralyzed muscles respond; the use of a faradic current when the paralyzed muscles do not respond, is actually harmful because contractions produced in neighboring healthy muscles may overstretch the paralyzed muscle groups.

Schaller of the Stanford University Medical School recommends that the electrical treatment be begun immediately following the acute stages of the disease, that is, after the temperature has subsided, the cerebrospinal fluid has cleared and muscular soreness has disappeared. He believes that electrical treatment is particularly indicated in these cases for two reasons: the first, to produce muscular contractions simulating normal movements and the second, to favorably influence nerve repair.

The choice of current to be applied is indicated by electrodiagnosis. When the reaction of degeneration is complete, he applies the galvanic current although, when interrupted, it has the disadvantage of producing considerable pain. The faradic and sinusoidal currents, while painless, probably have no more effect than the production of passive exercise to the affected muscles.

The affected limb should be well protected by ample coverings and the circulation encouraged by means of massage and manipulation. The application of artificial heat in such forms as hot

water bags or electric heating pads may be of value but heat must be applied with care as the skin is easily damaged.

In the secondary stage, as well as in the stage of final paralysis, a well applied mechanical support is of great advantage. It is important that whatever splint is used, it should be so applied as to keep the affected muscles well relaxed. The ingenuities of modern orthopedics have provided many forms of mechanical support that can only be referred to here; the details of these can be found in any standard work on orthopedics.

Tenotomies may be needed in order to restore function in instances where contractures have been allowed to take place. Such operations, however, will not be in order during the first two years of life and the same statement may be made in regard to nerve and tendon transplantation.

Epidemic encephalitis (encephalitis lethargica) is a disease that has made its reappearance throughout the world during the last few years. It is not an entirely new manifestation. It has been recognized periodically in Europe for several centuries. The first epidemic was described in 1720 and various other outbreaks have been reported, some in the eighteenth and some in the nineteenth centuries. Following the influenza pandemic in 1889 and 1890, isolated cases were described by Viennese physicians; eastern Europe seems to be a location where the disease is endemic.

Between the years 1890 and 1918 little was heard of this form of encephalitis, but following the world-wide visitation of influenza which occurred in 1918 and 1919, the reports of cases found their way into the literature. The earliest of these reports were issued in Austria. Very soon, however, Germany, France and England were visited, and later the typical cases of this inflammation of the central nervous system appeared in America.

The relationship of the disease to influenza is problematical. It is certain that cases do appear in the wake of an influenza epidemic, but whether the influenza is causative cannot be determined with certainty. The great degree of lowered nutrition in the populations of the countries that were at war and the lack of hygienic control in the passage of individuals from the infected areas have contributed to the sweep of the disease across the face of the globe.

It seems that the disease is due to a specific ultramicroscopic, filter-passing virus which finds lodgment in the nasopharynx of those affected. The onset of the disease is abrupt, followed by a

few days of malaise and fever. In many cases, this prodromal period is precedent to some days or weeks of increasing somnolence or lethargy. The appearance of oculomotor palsies is a striking, almost uniform feature of the visitation, although in infants, the somnolence and lethargy may be replaced by irritability, restlessness and urgent delirium, and the presence of oculomotor palsies is by no means always a uniform manifestation in affected babies. It may be very difficult to differentiate an acute meningitis from epidemic encephalitis of an infant patient. The final decision will rest upon the spinal fluid findings. The first sign that is most helpful in arriving at a differentiation, is the bulging of the fontanel. This occurs early in the case of meningitis. In epidemic encephalitis, the bulging happens relatively later and it does not often attain so marked a degree.

A fairly constant phenomenon of the disease in affected infants is the appearance of tremor, spasticity and isolated clonic spasms of the muscle groups. The condition of the reflexes varies with the area of the brain most damaged. If the pyramidal tract is chiefly involved, knee-jerks are increased, and there will be ankle clonus and a true Babinski sign. Neither the first signs nor the symptoms are of much aid in making the differentiation. On examination, if the spinal fluid reveals a lack of microorganisms and if the cell count is low, presumption in favor of epidemic encephalitis follows. The fluid is usually under a fair degree of pressure. The globulin test is positive, but precipitation of globulin is rarely extreme. These characteristics alone will not make it possible to identify a case of epidemic encephalitis, because the same characteristics are evident in the fluid of a tuberculous meningitis and it is not by any means always possible to detect Koch's bacillus in the tuberculous spinal fluid. However, these findings, taken with the history of acute onset and the rather rapid course of the disease and the absence of an increasing hydrocephalus, speak in favor of epidemic encephalitis.

The *treatment* of the disease is purely expectant. Young infants almost inevitably die through involvement of the medulla in the inflammatory process. In the second year the prognosis of the case is more favorable. Every effort must be made to maintain the nutrition of the child. Minor degrees of medullary involvement render swallowing difficult, and in such instances, gavage is essential. Evidences of mild involvement of the respiratory tract are best treated with atropin. This drug is the remedy of choice

when edema of the lungs is a complication. Lethargy and somnolence are best treated by daily application of the mustard pack. Good nursing must be insured. A quiet room, well ventilated and sunny, is of paramount importance.

The **spastic paralyses** of infancy may manifest themselves as either **monoplegia, hemiplegia, diplegia,** or **quadriplegia.** The commonest of them, **spastic paraplegia,** is generally known as **Little's disease,** although Little's original description was applied to a paraplegia in which mentality was fully maintained; in many cases, now diagnosed as Little's disease, there is definite mental deterioration.

The commonest causes of the brain damage which lead to this clinical manifestation are hemorrhage, antenatal or natal, and defects in cerebral development. A small number of spastic paraplegia cases can be traced to progressive degeneration of the brain, to a meningitis or to an encephalitis, especially when these affect the anterior and middle lobes. Rarely trauma or an infection may initiate the vascular changes which lead to the hemorrhage. There seems to be definite evidence that not all natal hemorrhages are due to trauma; there is reason to believe that many of them may be indices of the hemorrhagic diathesis of the newborn. In some instances the vascular changes induced by congenital syphilis have been etiologic.

The damage to the brain resulting from hemorrhage is often attributed to the use of the obstetrical forceps. While this instrument may be responsible for certain instances of intracranial bleeding, it is possible that many of these cases occur because the high forceps is not used soon enough. It is also possible that if Cesarean section were undertaken promptly by properly trained surgeons and in properly selected cases, that there would be fewer patients with spastic paraplegia.

During early infancy, unless the disability is very apparent it may be overlooked, especially as many of these infants are first-born children whose parents are unacquainted with the essential features of a healthy normal baby. But ordinarily, the general rigidity, especially of the legs, and the tendency to lie cross-legged in bed, will hold the attention of the young mother and she is apt to consult her medical adviser. In a considerable proportion of cases, postnatal convulsions supervene after 4 or 5 months; following a convulsive seizure, the condition of spasticity is noted. Perhaps it will not be until the time the mother expects the child

to begin to reach out for objects that the condition will be observed. Then it may be seen that the arms are spastic and that the hand is clenched and that the thumbs are held turned in. In such a child, especially when the spasticity is not of high degree, the disease may remain undiagnosed until the baby is first placed upon its feet. If the child is paraplegic, characteristic adductor spasm will be noted and the cross-legged attempts at progression observed. It will then be also seen that the calf muscles are contractured and a pes cavus or equinus has resulted. If the arms are affected, they will be extended, rigid and strongly pronated, the thumb folded into the palm and the fingers tightly flexed. The position of the hand and arm is strongly contrasted with that found in tetany in which the fingers are extended, the wrist flexed, and the forearm flexed in the semisupine position. Any or all of the body muscles may be rigid. Even the face muscles are sometimes involved. When the rigidity is not so extreme but that the leg may be flexed on the thigh, excessive knee jerks can be demonstrated. The Oppenheim and Babinski phenomena are readily demonstrated and ankle clonus is often extreme. Curiously, the abdominal reflexes in many cases remain normal.

As the spastic paralytic symptoms develop, the patient may show an athetosis; this is characterized by slow, recurrent, rotating, vermicular movements, which occur with or without intention. When these particular movements affect the face and the muscles of articulation, facial grimacing and speech alterations complicate the clinical picture. There are recorded instances in which the athetoid movements were the only evidences of cerebral damage and occurred apart from evidences of spasticity.

The spasticity of these cases is evidence of incompletely inhibited spinal control; older infants who have acquired a fair degree of ability to use their spastic limbs, temporarily become more spastic and awkward under the stress of excitement or emotion. Such a child who has clutched something in his grasping hand, may find it impossible to let go, or if he is attempting to walk, the sudden appearance of some one who arouses his delight or distaste, may bring on a condition of muscular rigidity that will render him entirely helpless, until after his emotion has passed.

A greater or less degree of mental defect is almost uniformly found accompanying spastic palsies. In many cases, the children are idiots; in others, the defect is evident only under excitement or stress. In a considerable number of cases, there is difficulty

in swallowing. Speech is commonly interfered with and some of the victims of this disorder are mutes. Blindness is less frequent and ocular muscle palsies are not very common.

There is no way in determining which of the cases of spastic paralysis fall into that group in which improvement may be expected, and which into the other smaller class in which the symptoms are progressive and for which nothing can be done. For this reason, treatment of all cases becomes important.

Undoubtedly, those in which the spasticity is slight yield more readily to *treatment,* but even cases that seem extreme, may show rapid and marked improvement under a proper therapeutic regime. The earlier the diagnosis is made and the treatment is begun, the more hope there is that amelioration will result. Of course a cure is never achieved; spasticity is never entirely overcome, but much lessening of spasm does occur and under careful treatment, a fair amount of muscular control is acquired through the use of the hot bath, massage and passive and active movements. Why these measures should be of so much value is hardly explicable when the pathology of the disease is considered.

The first bath of the course is given at about 100°. The first immersion is limited to 5 minutes. Each day, the bath water beginning at 100° is made ½° hotter when the child is in the tub, and the length of the child's stay in the tub is increased 1 minute. After about 2 weeks, most children will be able to endure an immersion of from 15 to 18 minutes at a temperature of about 108°; some may even tolerate 110° for part of the time. The object of the treatment is to secure temporarily, the greatest degree of muscular relaxation possible. This is best accomplished by the hot bath. The bath is to be followed by passive movement in order to discourage the full development of contractures. Massage is also given to overcome stiffness of the muscles and to aid in their nutrition. It is surprising how hot a bath a child can be trained to tolerate, provided the temperature of the bath water is increased not more than ½ of 1° each day. When the child is put into the tub, the temperature of the water should not exceed 100°. The increase in heat should be achieved by the careful addition of hot water through several minutes, and the bath maintained at the desired temperature in the same way. The relaxation after such a bath is positive; often the child is temporarily weak and somewhat exhausted.

While the child is in the relaxed state, which will last from

5 to 10 minutes, passive movements, alternating flexion and extension of the limbs at the joints, should be carried out rather rapidly, but not too vigorously. On completion of these movements, a thorough and complete massage of the muscles of the affected limbs should be given. When the trunk, as well as the limbs, is affected, passive movements to the spine should be undertaken.

Such a course of treatment extending over a period of time, (6 months to 2 years), will be of great advantage to the child, although it can never result in a positive cure. It is astonishing, however, to what degree restoration of function can be accomplished. The earlier the diagnosis of spastic paralysis is made and the sooner the bath treatment is initiated, the more satisfactory will be the recovery of function.

Tendon lengthening and stretching are important orthopedic contributions to the management of this disease. In selected cases where the spasticity is extreme, Forster's operation, which contemplates sectioning the second, third and fifth lumbar and second sacral nerves on both sides, may result in considerable relief, although many orthopedic authorities deprecate the procedure. It is, however, an operation which is rarely applicable during the first two years of life.

Spastic paraplegia with lost or diminished power in the legs, increased knee or ankle reflexes, sensory alterations and disturbances of sphincter control, occur whenever abnormal pressure is made on the spinal cord. During the second year of life, **caries of the vertebrae** is the commonest cause of such a clinical picture, although **tumors of the spine**, **pachymeningitis** and **hemorrhages** may be underlying causes. A radiogram of the spine may be of some value in identifying the site of a process in the vertebral column. However, to allow caries of the vertebræ to run on until compression and paraplegia ensue is indefensible. Tenderness and guarding of the spine, the pain referred, often to the belly, frequently to the chest or sometimes to the legs, should insure an early diagnosis.

The *treatment* of Pott's disease, if the case is diagnosed early, is hygienic and orthopedic. Fixation of the spine, removal of the weight of the head from the vertebral column by suitable apparatus or by recumbency on a Bradford frame, together with heliotherapy, hygienic and dietetic measures, useful for the treatment of tuberculosis in any other part of the body, are usually effective. (See chapter on Infectious Diseases.) In the more ad-

vanced cases of spinal caries, the operation of Hibbs or that of Albee will often prove brilliantly successful in relieving deformity and removing pressure from the cord.

Certain paralyses, the **obstetrical palsies,** are occasionally encountered. They are oftenest seen as a result of injury during delivery, to the nerves composing the brachial plexus. This may be unilateral or bilateral. Paralyses of the lower extremities, however, sometimes occur. The injury may range from a slight stretching of the nerves with a temporary cessation of function, to complete division of the structures. The degree of recovery depends upon the extent of the trauma. If the nerves have been stretched only, there is usually a complete restoration of function after a few weeks or months. On the other hand, if the structures have been divided with or without hemorrhage and a resulting cicatrization and there has been no coaptation of the broken ends, recovery cannot be anticipated.

The *treatment* consists of placing the arm in a sling or bandaging it to the chest in such a manner that the shoulder is pushed upward and firmly held in order that the broken ends of the nerves may be brought closer together. After two or three weeks of immobilization, heat and gentle massage may be employed to prevent muscular atrophy. Electrical treatments may be useful. If, after two or three months there has been no improvement, it is likely that the nerves have been completely divided and there has been no union of the broken ends. In such an instance, surgical interference should be considered.

There are certain very rare paralyses and ataxias which are generally classed as familial and hereditary. Of these, the best known are **Friedreich's ataxia,** a combined system degeneration of the spinal cord, **spinocerebellar ataxia,** resulting from a primary degeneration of the spinocerebellar tracts and **cerebellar ataxia,** in which the degeneration or lack of development is limited to the cerebellum. The symptoms of these conditions are incoordination of voluntary muscular movement and difficulty in maintaining equilibrium. Only the very earliest beginnings of these diseases will appear during the first 2 years of life. In the case of Friedreich's ataxia, the power to walk develops late and sometimes not at all. If it be acquired, after a time it becomes progressively worse and the child staggers but rarely falls. Attempting to grasp an object, the ataxia of the hands is at once evident. Articulation is defective. Optic involvement and mental deficiency are not usual, but they do occur in this disease.

Nystagmus is always a feature as are lateral curvature of the spine and pes cavus. As a rule, the knee jerks are absent, and the plantar response to stroking the sole is extensor. In the spinocerebellar and cerebellar ataxias, the recognized onset is usually later than the fifth year.

Although cerebellar ataxias are rarely recognized until the child attempts to walk, in a few recorded cases, due to failure of cerebellar development, tremor was the earliest symptom noted and this was seen shortly after birth. During the second six months of life, when the child should be attempting to sit up, it may be unable to maintain itself in the sitting position and if it is placed in this position and support is withdrawn, it immediately wavers and falls to one or the other side. Attempts to grasp objects offered it, show that the muscular movements are incoordinate. After a time, however, the child learns to sit up and to maintain itself in a sitting position, and eventually, to stand. When it does achieve the erect position, there is much unsteadiness of the legs, and the gait, when at last walking is mastered, is ataxic. The achievement of speech is delayed and when these children do learn to talk, it is with the slow, scanning speech characteristic of cerebellar disease. There is usually some impairment in the laryngeal constrictors and there is interference with swallowing. Intelligence is maintained and there is no nutritional disturbance, muscular wasting or loss of motor power. Muscle tone, however, is somewhat decreased, especially in the legs. The reflexes are unaltered.

Progressive improvement is the rule with congenital cerebellar ataxia, and by the time the child is 8 or 10 years of age, the symptoms will have largely disappeared. This improvement is synchronized with an assumption of cerebellar functions by an intact cerebrum. There is a form of cerebellar ataxia which does not go on to recovery in which the cerebrum is also damaged and is, therefore, unable to assume the functions of the cerebellum. Cases are on record of complete absence of the cerebellum, discovered at autopsy, in patients who showed no signs of cerebellar disease while they lived.

Occasionally, ataxia will come on acutely during the course of some illness and remain during the patient's convalescence only to disappear after a few weeks or months. Such seizures have been recorded as following measles, whooping cough, hemorrhages into the brain, convulsions and polioencephalomyelitis.

The **Werdnig-Hoffman** type of **progressive spinal muscular atrophy** is a degeneration of the spinal cord due to defective vital endurance. It is essentially a disease of infancy and the symptoms are of gradual onset and they may appear at any time during the first year of life. The most striking symptom is a gradual loss of muscular power. The child who has been sitting becomes unable to do so. The earliest weakness is noted in the pelvic girdle. The muscles of the back, shoulders, thighs, arms, fore-arms and legs are successively affected. Loss of the power of the proximal compared with the distal muscles is striking and long after the arms and legs are completely powerless, the fingers and toes can be moved with ease. Mentality remains unimpaired, sensation is unaltered and there is no pain, although it is a curious thing that very strong faradic currents produce no discomfort. These children are contented and happy. All the reflexes are lost, but although the muscles are in flaccid paralysis, the loss of tone is not so excessive as it is in most of the muscular dystrophies, especially amyotonia which has to be differentiated. In the later stages of the disease, bulbar symptoms supervene.

Congenital defects of the muscles are occasionally seen during infancy and in most cases are but of academic interest. Parts or all of the pectoral, trapezius and quadriceps muscles have been reported as absent, and as an extraordinary rarity, congenital absence of the abdominal muscles has been noted. Such children are unable to fix the chest cage and therefore they cannot cry or cough and usually they succumb early to some respiratory disease.

Myositis ossificans is another rarity that has been reported as early as the fifth month of life. The onset is with a localized swelling in one of the muscles of the back. It is firm, painful and the manifestation may be accompanied by fever. The skin over the swelling is not involved, although there is some subcuticular edema. This swelling tends to subside after a few days and fibrotic masses are left behind which eventually become ossified. As the disease progresses, the isolated bony masses become united and increase in size, sometimes uniting with neighboring bones and producing limitation of movement. The disease progresses through years, but during the stage of infancy, its only evidence will be the appearance of the swelling and the development of the small bone masses.

No effective treatment has been devised for this condition.

Certain children suffer from motor disabilities that arise from lack of muscle tone coupled with a slowly progressing atrophy of muscle groups. These **muscular dystrophies** or **progressive muscular atrophies** are rarely manifest at birth or during infancy, although in some instances from the time they are born, children of myopathic families may exhibit a flaccidity so extreme as to be incompatible with the continuance of life. Almost all cases of myopathy in which the diagnosis has been possible during the first year of life, have been examples of the **simple atrophic type of myopathy**, or of **myotonia congenita**—a clinical entity which many competent observers believe to belong in the group of simple atrophic myopathies. The very rare **Landouzy-Dejerine facio-scapulo-humeral myopathy** occasionally appears in early babyhood. When it does, the infant lies with a perfectly expressionless face, unable to modify the visage by contraction of the facial muscles. The scapulohumeral muscles are rarely involved until later. At first, there may be no more than inability to close the eyes or this inability may be joined with a curious loose thick-lipped flaccid and almost motionless mouth, so that whether the child be unhappy, angry, or glad, its facial expression remains unresponsive.

In **myatonia (Oppenheim's disease)**, or in simple atrophic muscular dystrophy, muscular disability is usually present from birth, but it may readily be overlooked until the child is some months of age when its failure to reach out, raise its head and kick its legs reveals the abnormality. As the condition varies in degree even at this time, slighter degrees of disability may escape observation, and only when the child should be beginning its attempts to walk is it discovered that its muscles are inefficient. Close scrutiny at this time will reveal that the muscles of the shoulder and hip girdle, although not in a state of atrophy, are lacking in tone and power. The disease progresses slowly. Oppenheim maintains that one characteristic of the cases which he has segregated and called myatonia is a progressive improvement; but the fact remains that no authentic case of cure or even of any striking restoration of function has been recorded in any case of this disease.

Among infants afflicted with this form of myopathy, there are the extreme cases in which all the muscles except those serving respiration are involved. The affected infant lies inert, unable to do much more than breathe. As time goes on, it becomes clear

that the intellect is unclouded. Such a child may live for a year or two to succumb finally to a respiratory disorder, or it may die within a few weeks or months from a similar complication. These extreme manifestations are seen in but a small proportion of the cases of this rare condition; when the affection is mild, the patient is able to move the head, but is unable to support it erect for more than a few consecutive moments. The limbs can be moved; provided no strain is put on them, but any attempt to support the weight of body results in an overpowering of the muscles. The bladder and bowel functions are maintained. It is demonstrable that the neural supply is unaffected, although some autopsies have revealed that the anterior horn cells, especially of the cervical and lumbar enlargements, were few in number and smaller than normal. Sensation is unaltered and intellection is high. During the second year, the child learns to talk and in mild cases, the power to stand erect or even to walk may be acquired, but almost all affected individuals remain unable to support themselves in the erect position.

The **pseudohypertrophic type of muscular dystrophy** is never seen in a fully developed stage until infancy is passed, but a history of those attacked almost always reveals that they were awkward in learning to walk and that from their first steps they fell easily and regained their feet with difficulty; therefore, when such manifestations appear, the possibility of a future pseudohypertrophic dystrophy must be kept in mind.

The *management* of the myopathies as they become evident during infancy, looks to the maintenance of muscle tone and to the prevention of contractures. These aims are best achieved by the daily use of massage, baths and passive movements. Drugs are useless and dietetic measures of no specific value. Contractures are unlikely to become so extreme during infancy that they demand tenotomy although possible necessity for this procedure must be kept in mind.

Interference with the **function** of one or more **cranial nerves** may be apparent from birth. It is sometimes unilateral. When the third nerve and the sixth are affected together, an ophthalmoplegia is found accompanying a ptosis; such a combination of disabilities, if coming on later in infancy suggests, especially if its development is gradual, the advent of a pontine tumor unless it is possible to relate the ocular weakness to a birth injury. However, such an ophthalmoplegia may be part of the picture of a meningitis.

An infant may be unable to move the eyes laterally; in order to see objects held to one or the other side, the head must be turned. Such a bilateral lack of power indicates an interference with the nuclei of the sixth nerves. When the symptoms are present from birth, it is usually because of a failure of these nuclei to develop. When the palsies are obviously the result of some postnatal event, they may represent an effect of meningitis, either inflammatory or serous in type. In the latter case, the palsy will be transient. Early in their course, pontine tumors may be productive of a unilateral strabismus indicative of involvement of the sixth nerve, but as an increase in intracranial pressure is also capable of producing this symptom, weakness of the external rectus is not uniformly a sign that permits accurate localization.

Loss of power in the masseter with consequent inability to suckle, indicates that the fifth nerve is involved as a congenital affection. It is hardly ever an isolated paresis but it happens accompanying a weakness of the muscular structures supplied by the motor branches of the seventh. Under these circumstances, the muscles of expression are inefficient, and it may be difficult to determine whether such an immobile face arises from nuclear palsy of the seventh nerve or whether it is the earliest expression of myopathy of the Landouzy-Dejerine type. The same symptoms arising in an older infant previously unaffected, should suggest damage involving the fifth and seventh nerves.

Involvement of the structures supplied by the seventh alone with resulting facial palsies may follow birth injuries due to forceps pressure. Usually the effects of such a mishap are transient unless, as may happen, the nerve has been severed. The path of this nerve as it runs in close apposition to the structures of the ear, renders it easily damaged in this part of its course. Middle ear disease, mastoid disease and periostitis without suppuration may give rise to a facial palsy. Fortunately, facial paresis of such origin is rarely permanent. Damage to the nucleus of the seventh nerve in the course of polioencephalitis may result in a transient or permanent facial weakness and irreparable paralysis may follow severance of the nerve during a mastoid operation or through separation of a sequestrum in the course of a neglected chronic infection of the mastoid.

In conjunction with congenital defects of the fifth and seventh nerves, weakness in the twelfth nerve also may be in evidence. In

such an instance, the tongue will be lacking in power and it will show some atrophy. One or both sides may be affected. As an acquired condition, these symptoms may appear during infancy as a result of tuberculosis of the bones at the base of the skull or they may result from the pressure of an exostosis. Tuberculosis about the pons or pontine tumors may also give rise to the same appearances.

It is rarely possible during infancy to diagnose with certainty involvements of the other cranial nerves affected by intracranial pathological conditions.

An enlargement of the head of an infant demonstrably due to an accumulation in the cerebrospinal fluid within the ventricles, constitute an internal **hydrocephalus.** It is the result of some interference with the normal course of secretion and reabsorption of cerebrospinal fluid.

The chorioid plexus is the specific secretory organ that elaborates the cerebrospinal fluid from the circulating blood. It pours its secretion into the ventricles which have little or no powers of absorption. As the fluid accumulates in these brain spaces, it finds its way into the subarachnoid spaces, particularly to those parts that lie at the base of the brain and are represented by the cisternæ.

When cerebrospinal fluid in excess occupies the space between the membranes and the surface of the brain, the result is an external hydrocephalus. This condition occurs only as a compensatory process succeeding atrophy or maldevelopment of the brain. It has little practical interest for it is rarely amenable to treatment.

Internal hydrocephalus may be caused by pathologic events that happened either before or after birth. In intrauterine life, the infections, especially syphilis, may originate the changes that lead to fluid accumulation. More often, in the congenital form, the pathologic state is one of defective or abnormal development of brain structures. The abnormalities interfere with communications along the complex ventricular drainage system and prevent the proper absorption by the circulation of the cerebrospinal fluid. In this group of cases, absorption is limited because access to the subarachnoid spaces is partially or completely hindered. A like interference to the passage of fluid from the ventricular system may follow an inflammation of the meninges. Whether this be antenatal or postnatal adhesions, proliferative changes

and distortions of passages may follow in the wake of acute inflammatory changes and dam up the secreted fluid behind them in the ventricular spaces. Within the ventricular system, lie the chorioid plexuses, continuously drawing cerebrospinal fluid from the blood and pouring it into the ventricles. As the ventricular linings have little or no powers of reabsorbing cerebrospinal fluid, any agency, even partly effective in blocking the outlets of the ventricles, must cause an accumulation of fluid, and this in a short time will give rise to demonstrable hydrocephalus.

This is not the only way, however, in which meningeal inflammations may cause hydrocephalus; many times after a bacterial invasion of the meninges, especially when the incidence of the infection is greatest about the base of the brain, matting and adhesions are left behind throughout a greater or less extent of the subarachnoid space. When such changes occur, the normal subarachnoid function of absorption is interfered with and an accumulation of fluid is the result.

These two sets of conditions both result in hydrocephalus, the one because the fluid is held in an area devoid of absorbing power, while at the same time its access to the subarachnoid space where absorption is possible, is obstructed; the other because the absorbing area, the subarachnoid space, is unable to function. On the basis of these anatomical differences, Blackfan and Dandy have proposed to classify **internal hydrocephalus** as **obstructive** and **communicating**. The two forms will occur in about equal proportion.

As signs of intracranial pressure, choking of the optic discs and Macewen's sign are apt to fail during infancy, but at this age, an increase in fluid content of the skull is usually evidenced by a widening of sutures and the development of a percussion note similar to that which may be elicited by tapping a ripe watermelon.

The history of the patient rarely helps the observer to diagnose the variety of hydrocephalus. To determine whether the morbid process is obstructive or communicating, he needs must have recourse to the use of certain tests. The technic of these tests is outlined in the chapter on Methods.

In an infant, a persistent increasing tension about the fontanel should be enough to arouse suspicion that a hydrocephalus is developing. Great enlargement of the head, and eye-ground

changes are late symptoms and when they have occurred, prognosis is bad. Spreading of the sutures, however, and re-opening of the lateral fontanels in an infant may be a fairly early sign. If the onset of hydrocephalus is acute and happens during the course of a meningitis, the earliest signs will be difficult to recognize.

Nuchal rigidity, head retraction, hyperactive reflexes, tremors, hyperesthesia coming on after a short period of slight abatement of the meningeal symptoms should put the clinician on guard.

Dandy found it possible to make ventriculograms of the hydrocephalic patient. He withdraws as much cerebrospinal fluid as possible by ventricular puncture, fills the spaces with air, and takes a radiogram. The resulting picture shows whether or not the ventricle is dilated and if it be, to what extent the brain tissues are damaged.

If a communicating hydrocephalus is demonstrated complicating a meningitis, a large proportion, (probably 75 per cent have this etiology) it is possible to aid the patient by *treatment.* The indication is for daily intraventricular puncture, the withdrawal of fluid and intensive treatment with antimeningococcic serum. Tapping of the cisterna magna (see Methods) and injection of serum at this site may well be employed. Forty-five c.c. to 60 c.c. should be injected after the withdrawal of a like amount of fluid and 15 c.c. to 45 c.c. daily, injected intravenously. After the meningeal inflammation has abated, the function of absorption by the subarachnoid spaces may become reestablished, at least in part. When a communicating hydrocephalus has become chronic, it is beyond medical aid. We have no effective therapeutic means of dealing with it.

If the tests prove the presence of a hydrocephalus due to an obstruction at the ventricular outlet and at the same time it is demonstrable that the subarachnoid absorbing system is damaged only slightly or not at all, surgical intervention is indicated. Whether the obstruction be due to an anatomical abnormality or is the result of adhesions after an inflammation, the callosal puncture of Anton will often provide a cure. In this operation, the skull is entered, the dura opened, a special instrument is passed down between the hemispheres, and an opening is made from the lateral ventricle to the cisternæ by breaking away some tissue at the lower internal wall of the ventricle, through and below the corpus callosum. Since Anton first described

this procedure, it has been successfully used by many surgeons in this country and in Europe. It is useful for the relief of the hydrocephalic symptoms, even when the hydrocephalus is subsequent to a brain tumor. The method was valuable when we had no exact means of differentiating obstructive from communicating types of hydrocephalus, so that now, when we can make the determination with accuracy, the callosal puncture procedure of Anton should prove to be of ever increasing usefulness.

It is rarely difficult to determine the presence of **mental deficiency** in infants, but in the early months of life it is almost impossible to establish the degree of deficiency. Therefore, the inclusion of a baby in the groups of **idiot, imbecile** and **feeble-minded** is impossible. The differentiation of these terms should be borne in mind. As given by the British Royal Commission on the Care and Control of the Feeble-minded, an **idiot** is "a person so deeply defective in mind from birth or from an early age, that he is unable to guard himself from the common physical dangers." An **imbecile** is a "person who is capable of guarding himself from such common physical dangers but who is incapable by reason of mental defect existing from birth or from an early age, of earning his own living." A **feeble-minded** person is "a person who is capable of earning his own living under favorable circumstances but who is incapable through mental defect existing from birth or from early age of (a) competing on equal terms with his normal fellows, (b) managing himself and his affairs with ordinary prudence."

Mental defectives may be divided into two groups: the one, primary, whose potential of development from the time of conception has been subnormal owing to defects within the germplasm; the other, secondary, which includes children conceived or born with a full normal potential for intellectual development but in whom disease has intervened to produce brain damage, anatomic or physiologic.

In the first group are included microcephaly, microcrania, Mongolism, congenital hydrocephalus and porencephalus, and congenital sclerosis of the brain. Individuals of the second group may have suffered from epilepsy, antenatal or postnatal, encephalitis or meningitis, postmeningitic hydrocephalus, cerebral degeneration, cretinism, nutritional defect, birth injury, or interference with sensory function (congenital mutism, congenital blindness).

It is extraordinary how often parents of a definitely defective child will state that it is intellectually acute. Therefore, it is

important before arriving at a decision about the mentality of a child, that the physician should give himself ample time to study the behavior of the infant and compare it with the behavior of other children of like age. If an infant under six months of age is under suspicion, it is well if possible to defer a statement which must include a prognosis, until the child is at least one year old. A second examination of the mentally defective baby, often profoundly modifies the impressions received at the first visit. The intellectual expressions of deficiency are only of relative value at this time of life, and it is on the physical signs and symptoms alone, that reliance can be placed.

The presence of gross physical abnormalities such as are seen in microcephalus, Mongolism and cretinism permit diagnosis of the actual deficiency of intellect to be made very early in life. Even the physical signs in a newborn of the Mongolian type of mental deficiency are so characteristic that there is no reason that the diagnosis should be overlooked. The appearance of the newborn cretin, in whom hypothyroidism is profound, is suggestive enough to arouse suspicion.

The persistent and frequent recurrence of **convulsions** without apparent cause in a baby during its first year of life, is a phenomenon that often indicates that the infant is suffering from maldevelopment of the brain and a primary type of mental defect. If the same type of recurrent convulsion should persist after an infant, previously normal, has recovered from an acute illness, the child should be carefully watched for other signs of a developing mental deficiency. The possibility that the patient is developing a hydrocephalus should be considered and one should search for the earliest signs of this disorder.

Not all convulsive seizures are of the major type. Attacks of *petit mal* occur either alone or together with the more severe convulsions. In young infants, the minor attacks are apt to be overlooked. They may consist of nothing more than a slight jerk of the head and shoulders or lower limbs, and a transitory loss of consciousness accompanied by a fixed expressionless stare that lasts often but a few seconds. These attacks may recur with extraordinary frequency. A history of 100 or more seizures a day is not uncommon.

The shape of the skull has significance in some cases, especially when the head is small and the measurements show the possibility of a microcrania. The mentally defective infant with **microcrania** and **microcephaly** usually has a sugar-loaf shaped

head. The premature closure of synostoses and cranial sutures is often a feature. Sometimes with premature closure of the fontanels, and sutures of the vertex, there is still possibility of growth from the bones of the base of the skull and the so-called "tower-skull" results. This deformity is not always, however, accompanied by a defect in the mentality.

A child may have a small ill-developed brain and a skull without striking malformation. In such a case, the space between the skull and the brain will be occupied by a cerebrospinal fluid and this state of affairs gives rise to the condition known as **external hydrocephalus.**

In the more profound states of mental defect, the child may lie perfectly immobile and be little more than a cardiorespiratory and digestive automaton. It is striking that such children exert so little muscular movement that their metabolic needs are little greater than their basal metabolism, with the result that they grow inordinately fat on a relatively low intake of food.

One of the most arresting signs of the less profound degrees of mental deficiency is the delay in the use of the voluntary muscles and in the performance of those simple motor activities which are the earliest signs of the child's orientation. Such children do not reach out to grasp objects until long after the normal child of the same age will be performing this act. Likewise, holding up the head, sitting erect, standing and walking are functions that are invariably much delayed in their appearance. The child too, may show delay in the use of its special senses in the appreciation of sounds or of brightly colored objects. The knowledge of the individual's environment is delayed and the child may not give a smile of recognition to nurse or mother until it is well past its first birthday; indeed, in extreme cases, these recognitions may never be possible. A complete inability to use language in a child who is obviously not deaf and who has passed its second year, is presumptive evidence of mental defect.

Infants who are intellectually abnormal are very likely to give evidence of their abnormality by aimlessness in movement, especially of the mouth, eyes and tongue. Hypertrophy of the tongue and its constant protrusion from the mouth is rather a characteristic sign of cretinism. Mongolian idiots are also likely to keep the tongue protruded constantly. Intractable crying without cause, in a child who is well and comfortably kept, should always suggest the possibility that the infant is mentally defec-

tive. Sometimes such children, when they reach the tenth or eleventh month, may begin to gabble incessantly, repeating unintelligible sounds from morning until night. Often this behavior delights the parents who have suspected the child's lack of mentality, and who now believe that this babbling is evidence of approaching normality of mind.

It is well to remember that certain mentally defective babies refuse to nurse. This refusal may be first evident just after birth or it may happen at any time during its nursing period.

The acquisition of proper control over the bladder and bowel especially the former, is delayed in mentally defective children and the dribbling of saliva, normal in early infancy, is a habit that persists.

The microcephalic child is distinguished by a narrow forehead, pointed vertex and flat occipital region. These peculiarities of the head and its small size render the appearance of the baby characteristic. As the child becomes older, the body usually grows at a greater proportionate rate than the head and the dwarfing of the cranium becomes more apparent. At birth, a normal child should have a head of 13½ inches in circumference; by the sixth month, the head should measure 16½ inches; at the end of the first year, 18 or 19 inches; and by the end of the second year 19 to 19½ inches. A child should be considered microcephalic, the circumference of whose head does not exceed 13½ inches at 6 months, 17 inches at 12 months and 17½ inches at 2 years.

The fontanel may be closed at birth. The palate is usually highly arched, while the ears are large and are usually deformed. The other features show no peculiarities. The child sits up, stands and walks late and speech is acquired later than normal and even then, the vocabulary is extremely limited.

During the second year, microcephalic children remain of the animal type—quick, active, restless, dirty and greedy. They show no response to affection. They often gain their amusement by destroying books and toys and tearing up papers.

About 10 per cent of mentally defective children show the physical characteristics which identify them as sufferers from the **Mongolian type** of mental deficiency. The term "Mongolian" was originated by Langdon Down. He was impressed by the Mongolian aspect of the eye; this similarity to the facies of the Mongolian races depends upon the characteristic fold of skin at

the inner canthus of the eye and the oblique direction taken by the palpebral fissures.

Nothing certain is known about the causation of this form of mental defect, but it is an impressive fact that a large proportion of the children so affected, are the first or last child of their parents, who often are over-young or are past the usual procreative age. Rachael Ash has pointed out that in many instances during the pregnancy, the mother was affected with some acute disease, most often an inflammation of the pelvic viscera.

The facies is easily recognized. All Mongolian idiots look as much alike as do the members of the race for which they are named. They have small, round, smooth heads, eyes closely set, usually behind oblique palpebral fissures. A well-developed canthic fold, present at the inner end of the palpebral fissure, is pathognomonic. Oculomotor defects are common and usually these appear as strabismus or nystagmus. The nose is small with a low bridge and a flat, wide negroid tip, with triangular nostrils. The palate is uniformly highly arched and narrow. As a result, nasal obstruction is frequent. Many Mongols are operated on for the removal of adenoids without result, because the obstruction is due to palatal deformity and not to an excessive amount of adenoid tissue. The average mentally defective child of the Mongolian type has an overlarge tongue which he delights in protruding and sucking. In the early months of life, except for its oversize, the tongue is normal in appearance. Toward the end of the first year, the papillæ become hypertrophied and the tongue assumes an appearance similar to that seen in the stage of desquamation of scarlet fever. During the second year, fissures develop across the hypertrophied epithelium of the tongue.

Except for the fact that the teeth are small, delayed in their appearance and subject to decay, they show no peculiarities. The hair is scanty, poorly nourished and dry. The skin is fine and in most cases without abnormality. In others, there is a well-marked hypertrophy of the follicles on the extensor surfaces of the arms. The ears are nearly always abnormal. They are round and small, and they tend to stand out from the head; in development, they are simian in character.

General muscular hypotonus is uniformly present. As a result, hyperextensibility at the joints is of significance in diagnosis and is of especial aid in helping to differentiate the Mongolian defective from the cretin. Further aid in this difficulty is to be had in considering the hand. The hand of the Mongol is short and

thick but it lacks the square, clubbed effect of the cretin hand; neither is the skin thick and wrinkled. The thumb and little finger of the Mongol are relatively shorter than the other fingers and the little finger in many cases has a peculiar in-curve deformity seen in no other condition. The hypotonicity extends to the fingers which can be pressed back until they touch the dorsum of the hand, usually without arousing any protest.

Mongols exhibit the same delay in the acquisition of voluntary muscular movements and normal bodily functions as do other mental defectives. They rarely learn to walk before their fourth year. They are feeble and clumsy in action and gait. They rarely learn to utter more than a syllable or two before the end of the second year. Convulsions are not more frequent in Mongols than they are in normal children. These patients are very subject to infections of the respiratory tract. Most of those who die in infancy, succumb to a bronchopneumonia. Those who pass their second year, often die in later childhood of tuberculosis.

In the infant *cretin,* the mental defect of the child is the result of hypothyroidism. See chapter on Internal Secretion.

There may be some question in the mind of the medical attendant who is confronted by a patient with a series of convulsions, **(epilepsy)** whether or not the convulsions are evidence of cerebral hypoplasia with mental defect, or whether mental defect is the result of cortical damage produced by the convulsion. True epileptic seizures occurring in infancy are prone to produce mental derangement. Dullness, apathy, excitability, accessions of anger and screaming, all are symptoms that may come on in a child who is subject to epileptic attacks. Fortunately, only a small proportion of the so-called "idiopathic" convulsions of little children are epileptic in character. Convulsions of unknown origin may occur many times a day, week after week, suddenly cease and leave only a temporary drowsiness and apathy, without permanent damage to the intellect. It is well known that spasmophilic infants may suffer for months and have daily seizures, and at the same time, exhibit very acute and active minds.

When spasmophilia, mental deficiency, structural defects of the brain can be excluded as causes of convulsions that persistently recur in an infant, then epilepsy must be considered as possible. When some or all of the attacks are of the *petit-mal* type in which the child is subject frequently to a momentary loss of consciousness, this possibility becomes all the more plausible. This plausibility becomes probability if a history of epilepsy

seizures occurs in the parents or in others of the child's family. When the attacks occur without apparent cause, in the absence of fever, infection, fatigue, great excitement or excessive emotion then, that they are epileptic in nature, becomes almost certain.

In the *treatment* of epileptic infants an unexciting regimen is of the utmost importance; ample sleep, monotonous surroundings, phlegmatic attendants, few visitors, simple bland diet with a limited supply of milk are the chief needs. Bromides are too frequently prescribed. They are of value immediately after an attack, in doses of 2 to 3 grains every 4 hours for a few days to a week. Luminal is quite effective in controlling the paroxysms, especially those of *petit mal*. Very small doses usually suffice. There is some advantage in combining the drug with calcium carbonate or lactate. For a 25 pound child, $\frac{1}{12}$ to $\frac{1}{8}$ of a grain of luminal with 3 grains of powdered calcium carbonate and 5 grains of sugar given morning and night will often control very severe cases. Larger doses may be given, but it is rarely advisable to exceed $\frac{1}{4}$ grain of luminal twice a day. The starvation treatment of epilepsy which has achieved a limited vogue is not applicable to the management of infant epileptics.

About one-half of the children who suffer from *spastic paralysis* whether this be diplegic or hemiplegic, show mental defects. These defects rarely are extreme in degree, although occasionally profound idiocy may be an accompaniment of a spastic diplegia. Patients with spastic diplegias definitely due to birth trauma are less apt to exhibit extreme mental defect than are others whose paralysis can be traced to defects in cerebral development.

Mental deficiency subsequent to attacks of *meningitis* and *encephalitis* is usually of extreme degree, although when the postmeningitic, pathologic process is a simple hydrocephalus, the mental abnormality may be of the slightest degree.

It is a moot question how far *congenital syphilis* is etiologic in producing mental defects as they are encountered during infancy. Undoubtedly, the effect of the poison of the Treponema pallidum can exercise such an influence over the germ-plasm, that there is interference with the proper development of cerebral tissues. It is especially through its influence on the lining of the blood vessels that the poison acts and is influential in certain cases of spastic hemiplegia and diplegia. It is, however, a fact that the incidence of syphilis on the cerebrospinal system is rarely made

evident before the fifth or sixth year of life, when juvenile paresis, progressive dementias and other forms of cerebral damage are met with. Congenital syphilis may be responsible for a low grade meningitis which may block one of the narrower foramina in the path of the ventricular outflow and produce a hydrocephalus accompanied by a mental defect.

There is a very rare disease in which the progressive development of mental deficiency is a striking feature. This disease is known as **amaurotic family idiocy.** Up to the present time, no authentic case has been reported which has occurred in an infant other than of the Hebrew race. The disease is really a progressive degeneration of the entire nervous system. The retina participates in the degeneration, and an examination of the eyeground shows the characteristic cherry-red area in the region of the macula. The disease makes its first appearance in a child who has been perfectly normal up to the fifth or sixth month. The first evidence is the development of muscular weakness. The child loses the power to sit erect and to hold its head up; it becomes apathetic, and it is obvious that the vision is impaired. Spasticity of the arms and legs develops, the knee jerks are exaggerated, and stroking of the sole results in the true spastic extensor response of the great toe. In the later stages of the disease, the knee jerk is lost and there is no response to plantar stimulation. The cerebrospinal fluid shows no alterations from the normal. Wasting and contractures develop in the later stages, and at this time the child becomes stuporous, has an occasional convulsion, is unable to nurse and inevitably dies.

The *treatment* of the mentally defective infant, in so far as it anticipates a cure, is hopeless. However, training and instruction in later infancy and in early childhood should be undertaken in order to develop the full intellectual potential of the affected child. For this reason, it is necessary that the infant as it develops should be kept under the observation of an interested physician and that every effort be made to observe and to utilize any educability that the child may possess and to develop his understanding of his environment to the highest degree possible.

From the earliest possible moment, attempts must be made to train the child in proper habits of life by developing regularity in his bowel function, improve his nutrition and in so far as possible, inculcate habits of cleanliness.

The food should be well chosen. It is to be remembered that

many of the older infants of this sort are unable or unwilling to chew; so that the proper preparation of their food becomes of great importance. In their second year, a great many mentally defective children suffer from the too exclusive use of a milk diet. If they cannot be induced to take solid or semisolid foods, cereal gruels and bread pap and vegetable pulps should be added to the milk in their nursing bottles. When they show evidences of malnutrition and rickets, then the addition of cod-liver oil and phosphorus to the dietary is of value.

Most mental defectives are subject to enfeebled circulation. For such children, tepid baths, cold douches, with vigorous rubbing, and the provision of adequate clothing are necessary.

Every attempt should be made to develop the mental potential of the defective infant. It should be sought to arouse, direct and hold his attention. During his second year, hardness and softness, heaviness and lightness, heat and cold, tastes and smells, should be impressed upon him. Find out what does arouse his attention and encourage him to concentrate upon it. Try to teach him to do things for himself. If he shows pleasure in some simple procedure, such as the shaking of a rattle, put it into his hand and shake the hand for him. It may take weeks before he will learn to reproduce the movement himself. But when he has learned to make even such a simple movement of his own volition, the cornerstone for a system of education has been laid.

It should be remembered that these children are prone to develop unpleasant habits such as thumb-sucking, dirt-eating and making disgusting noises, gestures and grimaces. Therefore, the very first beginnings of these tricks of manner should be observed and the repetition prevented. The struggle to prevent such developments may be a matter of many months, but the time is well spent.

Coercion and punishment are of no avail in dealing with the feeble-minded infant or child. Patience in teaching and development in the child of the sense of pleasure in accomplishment are the things that must be depended upon to make the very small beginning in infancy of a system, which in later childhood, may be used to develop the child's mind to the highest degree possible.

CHAPTER XVII

SKIN DISEASES

Most infants are born with **erythema.** The change of environment acts as an irritant and for some hours or days, the skin is reddened. Furthermore, the removal of the vernix caseosa which covers the skin of the newborn, exaggerates the irritation. The evacuations from the bladder and bowel are irritating to the newborn's skin, and as a result the buttocks become readily inflamed and they may become ulcerated. Unless great care is taken, cocci are encouraged and a dermatitis of the napkin area or an intertrigo, which is a dermatitis occurring within the skin folds, is set up. The evaporation of water on such a skin is especially irritating, and for this reason the greatest care must be taken after the bath to dry the baby thoroughly but without abrasion. Because of the delicacy of the epithelium, it is also important to avoid the use of soaps that are over-alkaline, whether these be used for the cleansing of the child or the laundering of the clothing, especially of the diapers. While the free use of a well prepared talcum powder is of great advantage, it must not be overlooked that some of the highly scented powders are very irritating to these skins and it is not uncommon to find an irritation due to their use.

The delicacy of the infant's skin and the fact that the child has not as yet acquired powers of immunity, render it especially susceptible to the lodgement of streptococci. As a result of such an infection, we are frequently confronted with the clinical condition known as **impetigo neonatorum (streptococcus impetigo).** This disease is characterized by the appearance of macules which pass through a transient papular stage and rapidly reach the condition of fully developed vesicles or pustules. Staphylococci become secondary invaders. It is not always possible to find the streptococcus in the lesions, although it is the originator of the trouble. The vesicles appear in rapid succession on neighboring parts of the body. They are most frequent and occur in the greatest numbers in the napkin region, especially about the groins, and in the axillæ, although no part of the body is immune from their visitation. In the napkin area and in the axillæ,

they are readily injured. The dermal covering breaks down and a certain amount of superficial ulceration with slight induration follows—a condition that gives rather a different picture than that seen elsewhere on the body in this disease, and for this reason a separate name, **vacciniform dermatitis** has been given to it. In the recently born, such an infection becomes a matter of some gravity. Although it is not always accompanied by fever, there may be a well-marked pyrexia or even a hyperpyrexia. As a complication, invasion of the blood stream may occur, with a resulting acute nephritis; sometimes such a streptococcic septicemia may result fatally.

In the more severe cases of **erythema,** not only do bullæ form, but there may be also a widespread dermatitis, exfoliative to a greater or less degree, upon which staphylococcus infection becomes engrafted. These complications give rise to a condition of general skin infection, most difficult to deal with successfully. Ritter described a few of the more severe cases of this sort in which the exfoliation was extreme and textbooks still refer to an analogous condition which is called **Ritter's disease.** Karl Leiner of Vienna also grouped some of the less severe of these cases that shed epithelium and gave them the resounding name **erythema desquamativa neonatorum.**

In older children, such impetigos are of less moment, although they may give rise to renal irritation. However, in such children, the distribution is rarely so wide and the eruptions usually are confined to the hands and face, and sometimes to the scalp. The disease rarely attacks the covered parts of the body. If it does, the crusted verrucous lesions of **ecthyma** are produced. It is always complicated by secondary infections and the formations of crusts which pile up and give the child an appearance unlike that caused by any other skin involvement.

The *treatment* of impetigos, in the simple forms of the disease as they occur in children who have passed the first month of life, offers no difficulties. The essence of the matter is to soften the crusts so that they will be readily removable and to apply appropriate antiseptic powders or lotions to the denuded skin that lies beneath. The use of a compress soaked in a 25 per cent solution of green soap, to which 5 per cent of salicylic acid may be added, will rapidly bring away the crusts. The application to the denuded areas of a 2 per cent ammoniated mercury ointment will usually effect a cure. If the crusts are not too bulky and ex-

tensive, the ointment alone will suffice to remove them and heal the underlying ulcerations. Some cases do not respond well to the ammoniated mercury ointment and for these, a calamine lotion containing 20 grains of aristol to the ounce will be found an effective remedy. However, mercury is the drug of choice, and if it happens as it does in some cases, that ointments are not well tolerated, the ammoniated mercury may be added to the calamine lotion and applied on compresses after the crusts have been removed by the soap compresses. Tincture of iodine is of value if it is applied to the lesions while they are in the macular or papular stage before they become vesicular. It is best not to apply the mercury and iodine at the same time because of the irritation that follows the combination of these drugs.

The treatment of the condition as it occurs during the newborn period, is rather different because of the tendency of the disease to spread more rapidly and because of its greater resistance. The first essential of treatment at this time of life is to keep all clothing and wraps away from the child's skin and to have it naked but surrounded by air warm enough to insure maintenance of body heat and at the same time to prevent perspiration. The arms are to be kept extended in order to prevent the contact of the opposing surfaces of the axillæ and so far as possible, the legs should be kept extended, so that the folds in the groin will not come into contact. On the tender skin of young infants, ointments seem to be less effective than they are in older children because of the maceration of the epithelium that they cause. Baths of 1 to 10,000 or 1 to 20,000 bichloride of mercury or of a ½ of 1 per cent cresol solution followed by the use of 1 per cent to 2 per cent ammoniated mercury in calamine lotion applied frequently to the affected areas, are effective; or the application of a compress soaked in a lotion made of equal parts of black wash, (B. P.), milk of magnesia and rose water with ½ per cent phenol, is often useful when the area to be treated is not too extensive. If the groins are badly affected, and it is found difficult to keep the folds from coming into contact, gauze compresses wrung out of this lotion and applied between the folds will accomplish the purpose and hasten recovery. As new macules appear, they should be treated by touching them at once with 5 per cent iodin in alcohol. In order to keep the child warm while it lies naked, a bed cradle, as pictured in the chapter on Methods, is advised. This can be kept at a desirable temperature

by the use of one of the modern electric heating pads, by the use of an electric globe protected by a wire cage and kept hanging in the cradle or where these are inaccessible, by the use of hot water bags, frequently changed.

Equally important, with the local treatment, is the maintenance of the highest possible nutrition and the provision of an adequate fluid intake. Great care must be taken to provide breast milk when this is available or failing this, a formula appropriate to the child's age, size and condition.

Beside streptococcus impetigo, infants may be subject to a widespread infection of the skin by staphylococci, **(staphylococcus impetigo)**. Such an invasion results in a clinical picture quite different from that of the usual types of impetigo. It is less frequently seen in the early weeks of life and usually attacks children already the subject of malnutrition. While uncleanliness and lack of care are apparent in many infant victims, the disease may occur among those most scrupulously cared for. Overclothing, with the resultant sweating and maceration of the skin, often plays a precursory rôle. Doubtless, unobserved infection on the fingers and in the nostrils of attendants, may initiate the infection.

The lesions consist of superficial, follicular spots of localized inflammation with thick whitish pus, rarely appearing as vesicles, together with a few or many simple furuncles. Usually these furuncles are not very deep although among the other lesions there may be one or two that include the deeper layers of the skin. On rare occasions, such deep boils may spread into small phlegmons which invade the subcutaneous tissues and give rise to the so-called "**subcutaneous multiple abscesses of infancy.**" The scalp over the back of the head is especially subject to invasion by crops of furuncles which lie at a moderate depth. Because the back of the head is so often hidden by close contact with the pillow, boils in this region may escape observation, especially in hospital practice. The cause of the distress of many a crying baby has been revealed as a developing furunculosis, when the occipital region was carefully examined. This is one reason why deep, soft pillows should be condemned, especially for babies who have malnutrition.

The *treatment* of these staphylococcic infections differs but little in principle from that of the other impetigos. It is an advantage to have the child unclothed in the warm cage pictured in the chapter on Methods.

One or two daily baths to which an antiseptic is added, followed by a thorough drying and dusting with an antiseptic powder, is all that is necessary for the slighter cases. A dram of aristol to one-half ounce of the stearate of zinc makes an excellent dusting powder for such an infant's skin. When the furuncles are developing, compresses of 25 per cent green soap hasten the evacuation of the pus and prevent the spreading of the infection around the boil because of the lytic action of the soap on the bacteria.

The use of the knife is advised only when the furuncles run on into cutaneous or subcutaneous abscesses. The absorption of the staphylococcic pus from these minute pockets is of small consequence while the incisions are painful and they do not heal as rapidly as do furuncles treated expectantly.

In furunculosis and staphylococcic impetigo of the scalp, shampoos with antiseptic soaps of which tar soaps are the choice, are of value. It is best to prepare a bowl of the suds and to lather the scalp well and afterward to wash off with a solution of boric acid.

There is no disease of childhood over which more controversy has occurred, than **infantile eczema.** The beliefs as to the etiology of the disease vary with the authorities who have considered it. At present, the view that it is an outward and visible sign of an inward and metabolic evil, is dominant. The truth of the matter probably is that eczema as we meet it in infancy is a symptomatic syndrome and there are several entities causing it, which our knowledge has not as yet allowed us to separate. Perhaps 10 per cent of all the cases can be classed as examples of skin lesions occurring together with the "exudative diatheses" first described by Czerny, and this 10 per cent it is that provides the small number of subsequent asthmatic children who have had eczema during their infancy. These patients are undoubtedly sensitized to some form of protein and the manifestations of the exudative diathesis are in truth, the revelations of an anaphylactic state. Sudden death is by no means an uncommon visitation among these individuals, and they die with symptoms identical with those shown by animals in anaphylactic shock.

The offending protein may be of bovine origin. The substitution of goat's milk for cow's milk in the dietary is sometimes helpful. Skin tests on the infant may reveal proteins found in the mother's dietary to which the child is susceptible. Shannon

has been successful in remedying the eczema by omitting such proteins from the mother's ration.

There is much to attract one to the idea that many of these inflammations of the skin that we call eczema, are the result of a postnatal continuation of the antenatal activities of the skin which in utero provided the vernix caseosa as a protection against the surrounding amniotic fluid.

An impetigo may be engrafted onto an eczema and the clinical picture will be complicated so that it is sometimes difficult to determine whether the impetigo or the eczema is the important lesion.

It should be clearly understood that the cure of an **infantile eczema of the facial type** is a matter of time. This type of eczema spontaneously clears up towards the end of the first year in contradistinction to the seborrheic type which tends to persist. There are some eczemas which, under the most faithfully followed treatment, will not entirely clear up; but even these can be kept sightly while the disease is running its course.

A protest must be made against the idea that the troubles of such babies can be alleviated by starvation. Nothing is sadder in pediatric practice than to see the starved, marasmic appearance of an infant who has been treated for weeks or months, in some cases, by the withdrawal of fats and in others by insufficient rations of carbohydrates or proteins; all this without the least effect on the skin other than to aggravate the lesion through the induced malnutrition. And nothing is more gratifying than to see the skin of such babies improve as their nutrition is increased under a properly calculated and balanced dietary.

The fundamental need of these skins is protection from air, water, mechanical trauma and infection. Protection against the air may be provided by various forms of masks in the severe cases and by the use of ointments and powders in the milder ones. A properly applied mask is also protection against mechanical trauma and dirt. (See illustration, in Methods.) The clearing away of crusts also is an instance in which the prevention of mechanical irritation is made effective. Scratching of the affected areas must be prevented and means of restraint of the hands is a practical device essential to the proper care of the case. Not only must the hands be restrained, but it must be made impossible for the child to roll over and traumatize the face by rubbing it on the bedding.

Water is to be kept away from the skin not because water or lotions are harmful, but because the evaporation of water on the skin is the most irritating single thing that can occur to an eczematous skin. As a matter of fact, wet compresses of boric acid or of dilute resorcin solution, are very effective in the treatment of the very acute stage of facial eczema, provided they are frequently changed and are not allowed to dry while on the skin. And it is possible to keep even an acutely inflamed eczematous skin clean by an occasional bath with dilute tar soap suds if only a very small portion of the skin is cleansed at one time, and this area is immediately dried after a preliminary quick rinsing with a boric acid solution.

The water used for baths is frequently treated by the addition of the gluten of bran. (For technic, see chapter on Methods.)

The use of the bath to remove crusts from the scalp is very important. A strong suds of tar soap well rubbed into the scalp and then washed off, is used. When the crusts are very dry and thick the stockinet mask, well soaked in petrolatum and left on overnight is usually effective. (See Methods.) Oil poultices are sometimes used but they are bulky and dirty and hard to hold in position. Sometimes several days' continuous use of the oily application is necessary to loosen the crusts. It is quite useless to attempt local treatment until all the dead epithelium and detritus is cleared away from the inflamed skin.

The management appropriate to a case of eczema, whether it be part of an exudative diathesis or an instance of simple infantile facial eczema, runs about as follows:

Use no water for cleansing of the face or the other inflamed areas.

Cleanse the inflamed skin with liquid petrolatum, light. (Benzoinol has proved in our hands to be one of the most effective forms of liquid petrolatum for this purpose.)

Whenever during the course of the disorder, there is oozing, weeping or cracking of the skin in the eczematous area, apply compresses of fine gauze soaked in a lotion composed of equal parts of black wash (B. P.), milk of magnesia and rose water, to which 5 drops of phenol or 30 drops of coal tar solution has been added. After a few hours' application of this lotion, the oozing should be controlled.

A stockinet mask (see Methods) soaked in melted yellow petrolatum (vaseline) is to be used to cover the scalp and face.

This mask serves first, to protect the skin from the air and from scratching, and second, as a bandage to retain the applications of ointment or the compresses of lotions close to the skin.

When there is much crusting and impetiginous complications, the free applications of 2 per cent ammoniated mercury or 3 per cent calomel, with ½ per cent phenol in cold cream, U. S. P., should be made. The ointment is to be spread liberally on the affected areas of the skin and the petrolatum-soaked mask applied.

The softening of the crusts should be followed by a single washing of the face with a weak solution of tar soap. The soap is to be rinsed off at once with a solution of boric acid. It is essential that only small areas of the inflamed skin be washed at one time and that they should immediately be dried by the application of soft muslin or old bird's eye pads. Care must be taken not to dry the skin in a way that will injure the repairing epithelium. The pads are to be pressed gently down on the washed parts and be lifted off without any rubbing.

When the crusts have come away, an ointment composed of ¼ to ⅓ per cent oil of cade or ½ per cent phenol, 2 per cent resorcin, in equal parts of Lassar's paste (no acid) and cold cream, U. S. P., is to be applied to the inflamed area and covered by the mask. A week's application of this ointment should abate the more acute phases of the inflammation.

After this, the mask may be removed and the same ointment applied to the skin. It may be well at this time to reduce the resorcin to 1 per cent strength.

In the defervescing stage, a calamine lotion or if the skin tends to be dry, a 2 per cent boric acid in lanolin may be sufficient and when itching persists at this stage, the addition of ½ of 1 per cent phenol is advantageous.

Pityriasis Capitis (milk crust, cradle cap) which often accompanies a facial eczema in infancy, but which sometimes occurs alone, is best treated by the free application of an ointment composed of precipitated sulphur and resorcin (5 per cent to 7 per cent each), made up in petrolatum. This ointment should be applied freely at night under a mask or cap. In the morning, a shampoo of tar soap-suds should be given and the soap immediately washed off with a saturated solution of boric acid. Care should be taken in shampooing and in rinsing the scalp, that no water is allowed to run down onto the face. It is important that the epithelial accumulations and scales be allowed to come

away by themselves. Combs and other mechanical aids are to be avoided.

When the lesions are dry and indolent, oil of cade (½ of 1 per cent), is sometimes a valuable addition to the resorcin (2 per cent), or calomel (3 per cent), or salicylic acid (4 per cent), ointment put up in lanolin and cold cream, (U. S. P.). The oil of cade stimulates epithelial regeneration and allays itching, although occasionally some skins react badly to its application. Under these circumstances, phenol (½ per cent) may be used to replace it.

When the eczema is obviously an expression of the exudative diatheses, atropin given internally in doses of 1/800 grain 3 or 4 times a day to a 15-pound child, is of value in controlling the vascular irritability.

Constipation must be combated. This can be done best by dietetic measures. Eczematous children need a properly balanced ration appropriate to their age and weight. It is of advantage to limit the milk ration; a pint of milk a day is as much as any eczematous child should get. The energy needs can be met by the addition of fats and carbohydrates.

Care must be taken that the child is not overheated, either by the temperature of the room or by the bed in which it lies.

Throughout the course of the treatment, the child's arms must be restrained by splints to prevent injury by scratching. (See Methods.) Another effective way of accomplishing the same purpose is to make a garment of fine muslin to be worn next to the skin. The sleeves of this garment should be cut about 3 inches longer than the length of the child's arms. The ends of the sleeves may then be pinned to the diaper. In this way, the arms are not entirely restrained, but the child is prevented from scratching.

Besides the ordinary eczemas of infancy, there is another type of skin lesion, usually classed as an eczema which reveals itself in infancy. The essential lesions of this disorder are brownish, indolent patches which appear often in the flexures of the arms and legs, and sometimes on various other parts of the body and on the face. They are usually subacute in intensity and persistent in nature. This is **seborrheic dermatitis** or as it is called **seborrheic eczema.**

For purposes of treatment, seborrheic dermatitis (seborrheic eczema), is to be sharply differentiated from the ordinary infan-

tile eczema, and from the skin lesions due to sensitization by foreign proteins.

The distribution of the lesions in the seborrheic type is characteristic in that they are found not only on the face but on the extremities, and body as well, as raised patches of more or less swollen corium, covered by brownish greasy epithelium which is chronically inflamed to a slight degree. Chronicity and indolence are characteristic of these patches. On the face, their distribution is not symmetrical, and although they do involve the cheeks, the tendency is for them to occur closer to the anterior margin of the ears and to run onto the postauricular skin. The flexures of the arms and legs are involved and in these regions the inflammation is, as a rule, a little more acute and much more persistent than elsewhere. The backs of the hands, especially about the thumbs, are frequent sites for this type of epithelial proliferation. Irritations may superimpose a more acute dermatitis onto these patches of so-called seborrhea and modify the clinical aspects of the case. The scalp, when it participates in the lesions, is covered in part or sometimes entirely, by a scurfy, sparse formation of scales which never attains the degree of accumulation that is characteristic of "milk crust."

This form of eczema is essentially a local disease of the skin. It has no proved relation to any metabolic disorder and makes no response to the many starvation or semistarvation diets which have been devised by dermatologists. As a matter of fact, children who are suffering from this disorder, do better on liberal than on restricted feeding.

It is not necessary to protect these skins from water to the degree advisable when we are dealing with the more acute types of eczema. As a matter of fact, in the indolent examples of seborrhea, applications of green soap and water, if they are allowed to remain on the skin about a half hour and then washed off, will often remove the resistant epithelial coating after a few daily applications. The result of this treatment permits such lotions and ointments as are useful in the treatment of acute eczema, to exercise their healing influence, promptly and effectively.

For the scalp in these cases, nothing is more valuable than a daily shampoo with tar soap followed by the application of a lotion containing spirits of turpentine, 20 minims; castor oil, 30

minims; oil of lavender, 20 minims; and enough 50 per cent alcohol to make 3 ounces.

As an application for the seborrheic patches, when they are slightly or moderately inflamed, a formula originated by George T. Elliot of New York has been more effective in our hands than any other. It consists of salicylic acid, ½ of 1 per cent; thigenol (or ichthyol), 1 per cent; tannic acid and zinc oxide, each, 3 per cent; all incorporated in cold cream, U. S. P. A little simple cerate may be added for pharmaceutical reasons. The salicylic acid contained in the combination is undoubtedly responsible for its efficacy.

It must be understood in dealing with these lesions, that no formula is applicable to every case of the disease. The amount of salicylic acid must be altered to meet the varying chronicity of the lesions. Very indolent lesions need sufficient proportions of the acid to be keratolytic while those in which the epithelium is less resistant, will need smaller amounts; or it may be necessary in some cases even to omit the drug for a time. It will not be possible to find a case in which all the widely distributed lesions are in the same pathologic condition at the same time; therefore, some patches will require soothing and others stimulating applications. For soothing, there is nothing better than the mixture, also devised by Elliot, composed of black wash, milk of magnesia and rose water in equal parts. Resorcin, if it is not used in a concentration greater than 1½ per cent or 2 per cent, and is combined with zinc oxide in unguentum aque rosæ and simple cerate, is also a soothing application when inflammation has supervened, either spontaneously or after salicylic acid stimulation. Occasionally, when salicylic acid does not stimulate these skins sufficiently, calomel in a 3 per cent or 4 per cent concentration may be applied and if the itching is extreme, coal tar solution, (liquor carbonis detergens), may be used together with the calomel ointment in the proportion of 1 dram to 2 ounces. However, for the most part, the salicylic acid-thigenol ointment will be effective. Failing a supply of thigenol, ichthyol may be used. The important thing about the use of any of these applications is that they should be closely applied, and this can only be done by making the application of the lotions or unguents on pads and bandaging these pads securely in apposition to the lesions. The mask may be used instead of the bandage when treating lesions on the face. Close application of the unguents to the lesions is

the first and most important point in treatment; the next is the careful avoidance of overstimulation. As soon as the evidences of slight irritation appear, recourse should be had to the use of soothing and protecting formulas and the treatment of the lesions becomes identical with that already outlined for the management of acute facial eczema.

Some authorities whose dictum is valued, have advised the internal administration of cincophen (atophan) in doses of 1 to 2 grains three times a day; in some cases, the use of this remedy seems to aid in clearing the skin both in the seborrheic and in the acute types of eczema. At other times, the use twice daily of $\frac{1}{10}$ to $\frac{1}{4}$ grain of calomel seems to be a distinct aid. Small doses of thyroid extract, $\frac{1}{12}$ to $\frac{1}{8}$ grains, 3 times a day, frequently hasten the clearing of the skin in seborrheic dermatitis especially if, as sometimes happens, there is an ichthyotic skin underlying the lesion.

In that condition known as **ichthyosis,** where the skin is thick, lacking in suppleness and subject to excessive epithelial proliferation and shedding, thyroid extract is also often of value but somewhat larger doses are required. In these cases, tar baths in the form of tar soap-suds and dilute solutions of coal tar (liquor carbonis detergens) seem of some value. After such a bath, it is important to dry the skin thoroughly and to anoint it with mineral oil. In this connection, it is important to note that both olive oil and mineral oil are very irritating to some skins and they cannot be used for every patient.

Urticaria is one of the commonest, most distressing and least understood of the skin lesions occurring in childhood. The analogy between the lesions of urticaria and those lesions that follow the injection of animal serums has led to the belief that urticarias are evidence of protein poisoning—an idea that is further borne out by the appearance of urticarial eruptions after the ingestion of certain foodstuffs, especially eggs, fish, strawberries, and raspberries.

Certain children develop urticaria persistently after the ingestion of orange juice. Others when chocolate is taken. During the period of the World War when the population was using wheat substitutes to a great degree, many children responded to the barley in their diets by urticarial lesions. What evidence there is seems to testify that it is the protein these foods carry that sensitizes the patient.

A few individuals are very sensitive to egg white. When this sensitiveness is extreme, the result is a state of affairs much more exaggerated than a simple attack of urticaria with wheals. Such children respond with giant urticaria—rapid and widespread edema of the face to such a degree that the eyes are closed. Occasionally the poison may be so profound that edema of the lungs and larynx supervene with great rapidity and a fatal termination may ensue before medical aid has time to reach the patient.

This sensitization is apparently a familial function in many cases and is found usually in more than one member of the family and in more than one generation. Fortunately these profound symptoms rapidly yield to the prompt use of sufficient doses of atropin especially if this be combined with adrenalin. To a child of 10 months suffering in this way from the giant urticaria of egg poisoning, 1/200 grain of atropin may be used at a single injection combined with 8 to 10 minims of a 1 to 1/1000 adrenalin chloride solution and injected into the triceps muscle. The prompt efficacy of atropin and adrenalin in this condition is further evidence supporting the view that these lesions are expressions of anaphylactic shock.

It is an easy matter when the child is found to be sensitive to egg white or any other protein, to immunize him to the poison by beginning the use of the given protein in minute dosage (1/1000 mg.) by mouth in capsule and to increase the daily amount ingested very slowly through 1 to 2 months until the sensitization is overcome.

Further evidence in support of the protein poison origin of the urticaria and of some of the erythemata is to be had from the fact that the bites of certain insects are followed by the appearance of widespread urticarial lesions over the body. These lesions, for the most part, are of the clinical type known as papular urticaria. This relation was pointed out as long ago as 1870 by Jonathan Hutchinson who also contended that pigmented urticarias result from bed-bug bites. In some regions of the Pacific Coast, during the flea season, papular urticaria is a constant source of irritation to the child and annoyance to the parents; but it is an annoyance that is much less frequent since the introduction of the vacuum cleaner which picks up the eggs of the flea so that they may be burned with the gathered dust. A few children are susceptible to mosquito bites and, apparently mosqui-

toes of certain localities are more virulent sensitizers than others. Ticks and spiders also on rare occasions are culpable.

It is possible that the widely held idea that sugars and starches are the causes of urticaria, may have some foundation. It is conceded that the growth of saccharolytic bacteria in the intestine following an excessive carbohydrate intake may produce acid enough to damage the epithelium of the gut and so permit the passage of unchanged protein into the blood stream. Such proteins might be derived from the ingested food or from the bodies of the vast mass of dead bacteria in the gut; for we know that when there is a sudden change in the reaction of the intestinal contents, many types of bacteria are immediately killed off in great numbers and their protein may be readily broken down and absorbed.

The ingestion of certain drugs, such as antipyrin or quinine, may give rise in rare instances to the appearance of urticaria.

If one accepts the protein sensitization theory and believes that urticaria inevitably arises from this cause, the rational method of treatment is either to eliminate the offending protein, or to immunize the patient. When insects can be incriminated, measures directed to preventing their attack on the child will be effective. Careful screening will prevent the activities of mosquitoes. Use of a vacuum cleaning apparatus which is within the reach of most city dwellers nowadays, will eliminate the flea.

The determination of what food is to be charged with the production of the urticaria when the wheals are of dietetic origin, is a matter of some difficulty and demands patience on the part of the child, the parents and the doctor. Certain foods that are known to warrant suspicion may be eliminated at first. The commonest of these is orange-juice, but there is no food that carries protein even in minute amounts that may not be the source of trouble. So that if the experiment of eliminating these more commonly causative agents among food stuffs is not successful, it is necessary to devise a dietary restricted to a single article of food such as milk, and to feed this alone for a few days and then to add, one by one, other simple articles such as wheat cereals, other less frequently used cereals, fruits and vegetables, allowing always 2 or 3 days to elapse before a new article of diet is added. Finally, when the offending food is found, it is wise to eliminate it for a week or two and then return to a second test. Now should it be found to be the undoubted causative agent, the child may be immunized

against it by feeding it in minute but increasing doses through a long period of time. The simplest method of accomplishing this is to begin by feeding a few drops of a weak decoction of the offending food. Little by little, more of the food is fed until the child can accept it in its usual form.

If there is reason to believe that bacterial proteins absorbed from the intestine are the sources of the symptoms, then the use of a purgative and the provision of a diet, well balanced but not so high in carbohydrates as to provide a very acid stool, will be of advantage to the patient.

Theoretically, the application of the skin test for proteins will aid in demonstrating to which proteins the patient is sensitive. Isolated proteins of different foodstuffs are obtainable. In order to test a child's sensitization, small amounts of one of these is placed upon superficial scratches made on the skin, usually of the forearm. The proteins are then moistened with a dilute decinormal solution of sodium hydrate and dissolved without traumatizing the skin. A positive response to the test is the production of urticaria-like wheal. There is some question as to the value of this test. It is certain that positive reactions to different foodstuffs occur in individuals who are able to ingest quantities of the same proteins without any apparent disturbance.

For immediate relief of the itching when the urticarial lesions are at their height, and the distress of the child is sufficient to keep it awake to the detriment of its general health, nothing is more effective than atropin and adrenalin as has already been suggested for the relief of giant urticaria. The dose however, need not be so large. One-three-hundredth (1/300 gr. to 1/400 gr. of atropin will suffice to make the lesion disappear in a few minutes and to entirely relieve the itching. When the lesions are less intense, local applications are all that is required. A lotion composed of phenol, 2 per cent, or coal tar solution, (liquor carbonis detergens), 3 per cent, in calamine lotion to which a little glycerin may be added, makes a very acceptable local application. Antipyrin by mouth has been highly recommended, but we have not had great success with it. English therapeutists have recommended an empirical remedy for urticaria which we have found of some value in those cases that do not respond to other treatment. It is a prescription containing the bichloride of mercury (1/200 to 1/500 gr.) to be administered by mouth.

All children with urticaria are made more comfortable by the

use of warm baths to which cresol, in proportion to 2 drams to about 10 gallons of water, has been added.

Some therapeutists prefer the use of ointments to lotions. For this purpose, a prescription containing coal tar solution, (liquor carbonis detergens), 30 minims; or oil of cade, 5 minims; in Lassar's paste (no acid) or unguentum aquæ rosæ, 1 ounce, makes an acceptable form of medication.

Miliaria (prickly heat) is an acute dermatitis which results from the irritation of perspiration, or of improper or excessive clothing or bedding. It is much aggravated by woolen bands and clothing. Itching and discomfort are features of this form of dermatitis. Babies affected with this, may suffer severely.

The *treatment* consists of frequent bathing with boric acid solution, followed by liberal applications of talcum powder. Attention to ventilation and appropriate clothing and the maintenance of proper temperature is essential.

Among the **parasites** which are causative of lesions on the skin in children under two years of age, the commonest are **pediculi, ringworm** and **scabies.**

Of the pediculi, **pediculus capitis** is the one usually found. Pediculus corporis or pubis, except among the lower class of immigrant population, is an extreme rarity. In Europe, pediculus pubis is responsible for a condition known as "pediculosis palpebrarum" in which the lice lay their eggs along the eye lashes and they are themselves found attached at the base of these hairs.

When infants under two years of age are attacked by pediculosis, needless to say other members of the family are also subject, and the treatment must be applied to all individuals who come in contact with the child. The clinical picture is modified by the fact that the skins of infants are so readily irritated that dermatitis and secondary infections of an impetiginous nature are easily superimposed upon the original irritation, so that the treatment already outlined for impetigo and dermatitis may have to be applied as part of the treatment for pediculosis.

Success in the *treatment* depends on detaching and destroying the eggs which cling to the hairs as well as on destroying the parasites themselves. The best routine measure is to rub onto the hair and scalp the tincture of larkspur. The hair is thoroughly drenched with this, the bedding protected by a towel wound around the child's head. After 12 hours, a shampoo, composed of vinegar and water, one to four, is applied in order

to loosen the eggs from the hairs. This is to be followed by a cleansing shampoo with dilute green soap or tar soap solution. This procedure is usually all that is necessary, but no treatment of the infant will be permanently successful unless the infested heads of the other children or adults in the family are sought out and the parasites eradicated.

The **treatment** of **pediculosis corporis** resolves itself into an alleviation of the irritating results that have followed the presence of the lice and a thorough search of all the clothing on the eradication of the insects by boiling or dry sterilizing. For **pediculosis pubis,** 2 per cent white precipitate ointment is effective.

Because of the peculiarities of the infant's skin, **scabies** in infancy does not assume the form of the disease characteristic as it occurs in older children and adults. The hands of the infant usually escape visitation and the characteristic burrows in the interdigital folds are lacking in many if not all instances. The more favored sites for the lesions in young infants are the folds of the groins and the axillæ. The face, rarely involved in later life, is almost always the location of some lesions in the case of an infant with scabies. In that class of society whose children are infrequently bathed, and in whom cleanliness is otherwise neglected, the secondary suppurative skin lesions, especially staphylococcic dermatitis and mild furunculosis may complicate the picture and make it easy to overlook the primary scabies.

Among the babies of the more enlightened classes who are kept very clean, scabies is not an infrequent gift from unclean nurses who have been allowed to take charge.

An infant's skin is so fine that the minute tunnels produced by the burrowing acarus may remain invisible; but as a reaction, there often appear small red itching papules. When these papules are present, they may be difficult to differentiate from the lesions of a papular urticaria. This is an error that ought to be guarded against. In all confusing cases a thin section of the epidermis should be removed with a sharp knife, and searched under the microscope for the acarus or its eggs. This procedure may be facilitated by adding a drop of sodium hydrate solution to the section on the cover glass.

Adequate *treatment* depends upon the fact that the acarus scabiei needs free access to the air. An ointment that effectively seals the openings of the burrows kills the parasite in a short time.

The addition of balsam of peru renders ointments more effective in sealing the burrows but this drug must be used with some caution as poisoning of susceptible individuals has been reported. One-half to 1 per cent of the balsam added to an ointment is as useful as larger quantities would be and will do no harm. The addition of sulphur is popular and effective and it has the added advantage of being antiseptic and curative to the secondary infection that almost always accompanies scabies in one part or another of the skin affected. It is important in young infants to use a washed sulphur in order that the sulphurous acids may not be in too high concentration; otherwise a sulphur dermatitis may ensue. Two per cent to 3 per cent washed sulphur, (precipitated sulphur), with 1 per cent balsam of peru in ordinary yellow petrolatum makes a good preparation. An ointment composed of 1 per cent or 2 per cent betanaphthol in petrolatum provides an effective treatment. The rubs with this salve, however, should not exceed 5 in number, and each should be preceded by a warm tub bath.

One of the commonest and sometimes the most persistent affections of the skin due to parasites, is the condition known as **ringworm**. The etiologic factor is a parasitic mould-fungus. More than 30 varieties of this parasite have been isolated. Some of these varieties attack only human beings although other forms affect animals as well. Therefore, cats and dogs may become carriers and transmit the infection to humans.

Ringworm affects the scalp and hair, the skin and the nails. When the scalp is involved, the condition of the hairs is diagnostic. The shaft of the hair breaks easily and the stumps, instead of being bristling as when the hair is closely cropped, are soft and pliable and are easily parted into furrows. The fungus begins its attack on the epithelium of the scalp but later it follows down the hair follicle and becomes firmly entrenched.

In cases where the diagnosis is doubtful, a few of the scales can be removed and dissolved in a little liquor potassae; by this procedure, the parasite can be demonstrated under the microscope.

For ringworm of the scalp, there is no satisfactory remedy except an effective depilation. Where a roentgen-ray machine is available, treatment in the hands of a radiographer capable of accurately measuring the dosage of x-ray, is the only rapid and thorough means of ridding the scalp of the hair. It is important that not only

the diseased hairs but also the healthy ones should be removed by the x-ray, although some criticism is always directed at the physician when a child is found to be bald following a treatment. However, after a period of about six weeks, the hair begins to grow and in a short time, it is as profuse as ever and often its texture and vigor is improved. Failing an accessible radiographic outfit and operator, or if the lesions are seen very early, the hair in the neighborhood may be cut short and then a fairly satisfactory depilation can be achieved by pressing adhesive plaster onto the lesion and pulling it off. The loosened hairs come away readily and following this attempt at depilation, ointments of oleate of mercury, zinc, nickel or eugallol may be worked into the lesion with a moderately stiff brush. A prescription containing nickel iodid 2 per cent, bichloride of mercury ½ of 1 per cent in benzoinated lard has proved useful in our hands. In the resulting inflammation, the parasites are often killed but an area of dermatitis is left; this has to be treated by soothing remedies such as a 5 per cent boric acid ointment.

Ringworm of the skin is usually amenable to the application of dilute iodin ointments. These ointments should be prepared in a penetrating base such as benzoinated lard and they should be applied after a preliminary application of green soap and a thorough lavage. When the lesions show some chronicity, it is of advantage to apply a 5 per cent salicylic acid ointment for 24 hours before beginning the use of iodin. Should the salicylic acid be followed by a dermatitic reaction, it is well to wait a second 24 hours before the iodin application is made.

Tenia versicolor sometimes appears on the skin of infants whose mother or attendant is also affected. Immersion after a daily cleansing bath in a ½ per cent to 1 per cent solution of sodium sulphite and the application of a 2 per cent resorcin ointment in a base of benzoinated lard is usually effective in eliminating this skin affection.

Pityriasis rosea, a lesion which is seen, but not very frequently, during the first 2 years of life, must be differentiated from ringworm, a lesion which it may closely resemble because of the peculiarity of its onset and the annular character of the lesion. There is always a revealing patch in this disease. This patch comes out some 3 or 4 weeks before the appearance of the many lesions which constitute the characteristic clinical manifestations. These lesions are usually smaller than the well developed lesions of

ringworm. They have a typical red color rather than the color of ringworm. There is no itching with pityriasis rosea. It is assumed that this disease is one of purely metabolic origin. It has a self-limited course, and the treatment resolves itself into a matter of diet and cleanliness. An antiseptic ointment such as is used in the treatment of ringworm, if mistakenly used in this disease, may aggravate the disorder and produce a dermatitis which is difficult to alleviate.

Toxic dermatitides occur with marked erythema, going on, in many instances to an exfoliation of the epidermis, which may be slight or extreme, according to the toxicity of the infecting agent, and the character of the child's skin. Such lesions are characteristic of certain ill-understood toxic agents which in our present state of knowledge, we believe to be of protein origin. Skin manifestations of the exanthematous diseases probably fall into this classification.

The **septic rashes** which are seen as a result of streptococcus invasion are certainly of this type. The erythematous prodromal rashes that appear from time to time before the onset of the true revealing lesion of variola, varicella, vaccina and measles, are also unquestionably of this general nature. The rashes that result from the intake of serums, we also class with these toxic dermatitides. The erythrodermias of the newborn, (Ritter's disease and Leiner's disease) are now known to be the end results of septic erythemata, due probably to streptococci. This may be true also of the **recurrent desquamative erythema** first described by Osler.

Analogous erythematas with desquamation follow the use of **antipyrin, quinin, bromides, the salicylates and belladonna. Poison ivy, poison oak** and **primrose** are sometimes offending agents and produce a skin lesion of distressing character. They are best treated by gently washing the affected parts with soap and water, drying with alcohol and applying a lotion composed of 1 per cent phenol, ¼ of 1 per cent aluminum acetate, 2 per cent aristol, and 4 per cent zinc carbonate in calamine lotion.

Among the less severe and more transient erythemata, there are many which apparently arise from the ingestion of *food proteins* or from some bacterial poisons absorbed from the intestine. These are probably akin to the milder forms of urticaria. Especially of interest to the physician dealing with children are the erythemas that sometimes follow the use of an enema containing *soap.* Whether these result from the absorption of the soap or from some protein body included with the variety of soap used,

or are the results of bacterial products absorbed from the intestine which has been rendered permeable by irritation from the soap of the enema, one can never say; but the clinical fact remains that the infant may develop so extensive an erythema as to alarm the parents and even to disturb the equanimity of the attending physician unless he is familiar with this possibility. Such transient erythemata are self-limited and need no treatment.

Pemphigus, an uncommon disease in childhood, is of unknown origin. It must not be confused with the condition known as pemphigus neonatorum which most often is, in reality, an impetigo, or else an evidence of congenital syphilis. The lesions consist of blebs on the skin, each possibly as much as 3 or 4 inches in diameter. These blisters become turbid and rupture after a few days and healing occurs without scarring. There is little or no constitutional reaction, unless the lesions become infected. The streptococcus is a dangerous invader and pains must be taken to protect the skin from contamination. There is a tendency to relapse but the prognosis is good. The use of local antiseptic washes is indicated after the rupture of the blebs and dietetic and hygienic measures are in order to raise the resistance of the infant.

Psoriasis is a rare affection of the skins of infants although it is not uncommon in childhood. It is characterized by circular patches of silvery scales that grow by extension at their margins; these scales can readily be scraped off and the skin underneath shows no inflammatory change. The disease is not contagious so far as is known and it has a tendency spontaneously to clear up and to recur at short intervals. *Treatment* consists in the removal of the scales by the application of a mild salicylic acid ointment (3 per cent), or by a vigorous rubbing with tar soaps or with dilute coal tar solution. A simple method of treatment, often very effective, is to paint the areas with collodion. Two to 4 per cent chrysarobin may be added, if desired. The quartz lamp is reported to be effective and may be used in resistant cases.

Nevi (birthmarks) are congenital skin blemishes. Their cause is unknown. The most disfiguring variety is the vascular nevus or congenital angioma. Nevi may appear on any part of the body and they are most commonly seen about the face, neck and scalp. The most effective *treatment* is the application of carbon dioxide snow or the use of radium; these procedures are best entrusted to the skilled dermatologist.

Moles are composed of pigmented thickened epithelial growths from which coarse hairs spring later in life. They are harmless but disfiguring, although a few have been known to take on a malignant character during the later decades of life. They can be readily removed by electrolysis; however, this is rather a painful process. The simplest method for their removal is to cut a piece of carbon-dioxid snow about the size of the blemish and apply it for thirty to sixty seconds. A blister forms and the mole comes away and on healing there is but a slight scar.

Verrucæ (warts) are quite common in childhood although not often occurring in the first 2 years of life. They are epithelial growths, doubtless the result of microbic irritation, although the infecting agent is as yet undiscovered. They are infectious and autoinoculable and have a peculiar tendency to come and go, a characteristic which accounts for the many lay wart cures. Destruction of the growth by glacial acetic acid or by nitric acid is inadvisable as the method is painful and any excess of acid may damage the surrounding skin. X-ray exposure is effective. (See chapter on Drugs.)

Pellagra is a constitutional disease, with skin manifestations, rare during infancy, although an increasing number of cases are being reported. The cause of the disease is unknown, but there are many authorities who believe that the disorder is one of the food deficiencies. However, it must not be forgotten that there are a number of cases on record where the babies have been fed exclusively at the breast of robust, well-nourished mothers whose milk had been proved to be of normal composition. The disease appears most often in rural districts and in localities where sanitary conditions are bad.

The affection manifests itself by symptoms referable to the gastrointestinal, cutaneous and the nervous systems. Disturbances of digestion are invariably present. There is either a severe diarrhea or an obstinate constipation. Skin involvement becomes apparent early in the course of the disease as a symmetrical, erythematous rash, distributed over the dorsal surfaces of the hands, wrists and feet, and on the ankles. Sometimes the forearms are affected and very often the rash appears on the neck and the forehead. A symmetrical erythematous rash of this sort is stated to be pathognomonic of pellagra, if it occurs in combination with severe diarrhea or persistent constipation. In the course of the disease, some nervous symptoms are certain to develop. The commonest of these are parasthesia and insomnia.

Attacks of transient muscular rigidity may occur, and the reflexes, especially the knee-jerks, become exaggerated. A study of the blood will reveal nothing helpful to diagnosis.

For the *treatment* of the irritations of the erythematous skin, applications of calamine lotion are useful. The disease itself responds to hygienic measures which consist of a complete change of environment and of diet. The child, if a nursling, should be removed from its mother's breast and supplied with the milk of another woman, one known to be free from pellagra. An older infant or one who must be artificially fed, should be given a liberal diet that will supply its energy requirements and which carries an abundance of both fat soluble and water soluble vitamines.

Acrodynia (erythredema, dermatopolyneuritis), first reported by an Australian observer, Swift, was brought to the attention of the medical profession by Bilderback of Portland, Oregon. The disease occurs with moderate frequency, attacking older infants and young children.

The clinical picture is one which gives evidence of changes in cutaneous, nervous, dental and gastrointestinal tissues. There are also striking temperamental, psychic and sensory changes together with an assumption of odd postures.

The earliest signs of the malady are irritability, restlessness and emotional instability. These are soon followed by disturbed sleep, profuse sweating and anorexia. Abdominal pain may be persistent, and accompanied either by constipation or by diarrhea. The latter symptom can be so pronounced that it will produce an alarming dehydration. The looseness of the bowels is aggravated by milk in the diet and ameliorated by generous rations of green vegetables, fruits, and digestible carbohydrate.

Soon after the appearance of nervous symptoms, the skin will show effects of the disease. Usually, an intense dactylitis-like swelling precedes the eruption on the toes and fingers; as well, a swelling of the palms, soles and dorsal aspects of the hands and feet appears, and at the same time the extremities become slightly cyanotic or, more often, erythematous. A little later vesiculation of the fingers, toes, and palms appears; this in turn is followed by a worm-eaten appearing desquamation of these parts. Some erythema and papular indurations of the skin of the body occurs in every case. In certain, however, these changes are intense and so extensive as to leave but little of the skin uneffected.

At this time analogous lesions appear in the mucous membrane

of the mouth, producing a stomatitis and gingivitis. The gingivitis may be proliferative and the deciduous teeth be shed because of it.

Photophobia, paresthesias and itching together with tenderness of the mouth combine to make this disease one of the most unpleasant that sick children have to endure. Unless the hands are restrained, scratching becomes incessant and secondary infections give rise to impetigo or furunculosis from which septicemias and osteomyelitis have been known to arise.

The assumption of queer postures is very striking. The commonest is the knee-chest position with the legs drawn up. Muscular hypotonicity which is a feature of the disease permits many peculiar attitudes to be assumed: as, for instance, one child may lie on its bed with thighs fully flexed and the toes pointed over the shoulders, while another may lie with its head so hypoextended that the forehead presses against the bars of the crib. Apathy, dullness and listlessness, so dominant when the child is left to itself, give way to restlessness and irritability if he is interfered with.

A large proportion, but by no means all, of the children have upper respiratory tract infection; in some persistent severe bronchitis of the middle and larger tubes is a feature. About half suffer with constipation, and half with marked diarrhea. A number of the girl children at some time or another in the disease, have a great deal of pus in the urine.

As the etiology is unknown, the *treatment* must be symptomatic. It includes rest in bed, fresh air, exposure to the sun, and forced feeding. Appropriate treatment of infected tonsils and sinuses, and of other complications must be thorough. The diet should be a liberal one from which milk is excluded. For the sweating, atropin is useful. Stomatitis and gingivitis are improved by scrupulous cleanliness and helped by a spray of zinc chloride lotion (see formula on page 635). Persistent sleeplessness can be overcome by the use of chloral hydrate in doses appropriate to the weight of the child.

A dusting powder containing phenol, calomel, bicarbonate of magnesia together with zinc sterate effectively controls the itching, especially when the eruption is in the vesicular stage. Applications of calamine lotion are also useful. Cod-liver oil sometimes seems to act as a specific and at other times it is without effect. The disease is self-limited and does not tend to recur, once it has entirely disappeared.

CHAPTER XVIII

GENITOURINARY DISEASES

The urine passed by the newborn is pale and clear and it may be in considerable volume. It is the result of prenatal secretion and contains little urea, relatively much uric acid and traces of albumin. After birth, at first the urine is scant, sometimes even wanting for 24 hours or longer. During the first week of life, it is dark in color because of bile and broken-down blood pigments that are being excreted. After this period, there is a change.

The volume of the excretion is variable but increases during the first year. Careful collection shows that the child should pass anywhere from 10 to 20 ounces in 24 hours progressively from the end of the first month to the end of the twelfth. The specific gravity of the urine in infancy is low and rarely reaches above 1,012; usually it is below 1,010. The reaction is acid especially in the first 2 weeks of life. During this 2 weeks the urine may vary in its composition because the processes of metabolism are intensified from those of the prenatal period and the kidney has new work to do which is evidenced in the frequent presence of albumin and a slight granular deposit on the diaper composed of uric acid, urates and sometimes oxalates. The presence of casts at this time is of no importance unless there be a concomitant infection. In prenatal life, there is apparently some albuminous material accumulated in the tubules of the kidney which is washed out and excreted during the early postnatal days as casts. These have some interest as it is now known that they form a matrix in which is deposited ammonium urate and uric acid with the end result that the so-called "uric acid infarcts" of the kidney are built up.

When these infarcts appear in the urine, they are found during the first week though rarely before the end of the first 48 hours. Their passage occasionally damages the urethra and may be the cause of a mild hematuria, very alarming to the attendants. Occasionally, such an infarct may lodge in the deep urethra of a boy and cause a great deal of distress from a distended bladder, with pain expressed by intractable screaming. However, in-

creasing cystic pressure will almost invariably dislodge such an obstruction. While it is generally held that the period of uric acid excretion is limited to the first week or ten days of life, some children continue to be troubled by the passage of these concretions during the first 3 or 4 months; the writers feel that many children who are under constant treatment for what is considered to be colic, are in reality suffering from the distress incident to the passage of ammonium urate or uric acid in the form of sand or small concretions. Some of these masses appearing on the napkins are softened, absorbed by the fabric, and they show as small spots of bright red color, being frequently mistaken by parents or nurses for blood stains—a perfectly excusable error.

Frequency of urination varies with different children and with the amount of fluid intake. The voluntary bladder control begins to be expressed toward the end of the first year and by the end of the second year, the voluntary should entirely replace the involuntary guidance of the bladder. **Enuresis** in older children, when there is no definite condition that produces irritation of the genitourinary tract, is essentially an expression of a maintained involuntary as opposed to a voluntary control. It means either that the child has never had the cerebral mastery of the pelvic viscera properly established, or that through bad environment and imperfect training, a neurotic habit has been encouraged; this happens even after cerebral control has been attained, with a reversion to the infantile involuntary type of bladder evacuation without central inhibition.

Insistence on proper urinary habits during the second year of life will almost invariably act as a preventive to the appearance of enuresis in later childhood. Of course, it is hopeless to expect the type of parent who is himself so neurotic that he cannot bear to impose any inhibition on his child, so to discipline and train the offspring that the child will acquire control of the urinary function.

Every willed evacuation of the bladder tends to break down the lower spinal reflex and after a few weeks or months of training, the involuntary micturition is completely replaced by a voluntary act. The whole technic of this training depends upon success in anticipating involuntary urination by a voluntary effort. In those children who have the habit of hourly urination, it will be necessary for a few days to induce them to empty the

bladder every half-hour, increasing the interval between the willed urinations little by little until a normal habit is acquired. It is especially important, if such children are bed-wetters, that they be taken up during the night at intervals which will anticipate the usual times of their bed-wetting. This procedure is perfectly useless, however, unless the child is thoroughly wakened and induced to perform the urination voluntarily. If the child to be treated is neurotic and of a neurotic family, such a treatment must be entrusted to a placid and competent trained nurse or to some other competent attendant and not be left to the family.

It is better if the fluid intake be restricted after 3 or 4 o'clock in the afternoon. A little pains will usually convince the parents that a child cannot possibly suffer from thirst during such a short interval and water drinking at that time is usually a habit acquired during earlier infancy from nursing or taking a bottle on going to bed.

The mutilation of children, circumcision of boys and ablation of the clitoris in girls in order to relieve enuresis, is certainly indefensible. The ablation of the clitoris in order to relieve an enuresis is barbarous. Most of the circumcisions done to achieve the same end are equally deplorable; but it does happen occasionally that a boy has so tight a phimosis or so much retained smegma that the operation is thoroughly justified. But circumcision can be no more than an adjunct; of itself, it cannot produce a cure of enuresis.

The irritations of concentrated or highly acid or unduly alkaline urines must be corrected. Diets which provide excessive amounts of fluid must be rationalized and the nervous system of a child quieted or stimulated as the indication may be. Certain children have a highly exalted sensorium and apparently the sphincters of their bladders relax with greater ease and allow a few drops of urine to initiate the involuntary reflex which empties the viscus. If any drug is indicated in such children, it would be belladonna or the bromides or a combination of both. If it is a nocturnal enuresis that is troubling, the simple expedient of a board under the mattress and the elevation of the foot of the crib on 3 inch blocks is occasionally effective by purely mechanical means; it keeps the urine away from the neck of the bladder. In this class of patient too, it is important to know that there are no other sources of pelvic irritation—that the lower bowel is

properly evacuated, for rectal constipation is often a result of an unstable nervous system. Occasionally, irritation may arise from the presence of oxyurides and the enuresis will not yield until the worms have been eradicated.

In another type of child, anemia, low muscular tone, and a mild degree of malnutrition seem to be the underlying factors favoring enuresis rather than any infirmity or instability of the nervous system; in such cases, the correction of the diet and improvement of nutrition together with the use of tonics, such as nux vomica with iron will often produce a result that is most gratifying.

The presence of casts and of cellular elements in the urine of infants without other clinical signs is not necessarily an evidence of true **nephritis**, nor does the appearance in the urine of a few streptococci, pneumococci or colon bacilli testify to the presence of an infectious nephritis. The colon bacillus will usually be found in any specimen of urine that has not been obtained in a female under the strictest aseptic precautions by catheter; its presence without symptoms is not significant. In infection of the tonsils, pharynx and nasopharynx, so common during this period of life; in the blood stream infections, more usual than is generally thought; in lesions of the skin, such as impetigo and furunculosis; in true bacterial infections of the gastrointestinal tract; in all these, almost inevitably, casts, white blood cells and pathologic bacteria will be found in the urine. The presence of these bacteria in the urinary secretion indicates definite renal cell injury, which, on the one hand, may be so slight that repair speedily follows, or on the other, so grave that a recognizable infectious nephritis is developed.

The types of chronic nephritis encountered in older children and in adults, are almost unknown during the first years of life. The characteristic kidney inflammation of infancy is **acute hemorrhagic** or **glomerular nephritis**. Subacute, **tubular nephritis** may follow the glomerular variety but among well-cared-for children, under proper medical supervision, it is very rare in this country. Acute glomerular nephritis must then be looked upon as a renal irritative expression of a bacteremia revealed by a decrease in the urinary output, by the presence of red blood cells in large numbers, and by a certain amount of toxemia referable to a decrease in the excretory function of the kidney.

It is chiefly water excretion that suffers at first thorough dam-

age to the glomerular structures, which are the essential excretory organs for water in the blood. When interference with this function becomes profound, water may accumulate in the tissues and edema supervene.

Fever, which is usual in these cases, results probably from two factors. It is certain that the reaction to the underlying infection is causative, and it is probable that alterations in water metabolism following an accumulation of mineral salts in the body also play a part.

Edema which occurs so frequently in infancy as a result of metabolic disturbance, taken alone, can never be regarded as sufficient evidence to warrant the diagnosis of a kidney lesion. It is much more apt to have come from a metabolic disturbance due to a dietary insufficiency or to protein sensitization, than it is to have arisen from impaired renal function.

As **acute glomerular nephritis** is essentially a disease of the filters of the kidney and as the principle of resting a diseased organ so far as possible, is generally accepted as valid, it appears that the logical treatment of a nephritis of this type should call for a limitation of fluid intake to the least volume compatible with the maintenance of a proper circulation in the blood vessels. Further, it is logical in devising a diet to meet a nephritis to attempt to relieve the kidneys as much as possible from excretion of waste products. As the waste products excreted by the kidneys are chiefly derived from proteins, there can be little objection to the limitation of protein except when there is evidence that the child is suffering from a low protein content of the blood—a state of affairs which is unlikely to happen in conjunction with the early stages of acute nephritis.

In practice, the application of these principles results in a diet for temporary use in which water and milk are limited and in which fats and carbohydrates form the basis for nutrition. Fortunately, in the first 8 or 10 months of life, glomerular nephritis is unusual, although it does occur even during the first month. At this age, fluid restriction is very difficult to maintain, even for a short period of time. The nearest that we can attain to it in practice is by the use of thick formulas made with the addition of cream, instead of with skimmed milk as is outlined in the chapter on Vomiting. When such a child is breast-fed, the limitation must be exercised through a decrease in the number of meals and in the length of the nursings. For older infants, during the first 2 or 3 days of treatment, fluid can be limited to 8 or 10 ounces divided

into small doses and given at 1- or 2-hour intervals during the 24 hours. Fruit sauces with cream, concentrated cereals with butter and sugar or when the children are old enough to eat it, softened toast or crackers with butter or jelly, provide the main elements of the diet. After 48 hours on this rigidly low fluid intake, 3 or 4 ounces more of water per day may be added for a few days and then another 3 or 4 ounces, although it is astonishing with what comfort children from 10 months to 2 years of age will endure this restriction of fluid. In children beyond the nursing age the addition of salt to the food should be interdicted. The promptness with which an edema will often disappear following the withdrawal of sodium chloride from the diet is very gratifying.

Water evacuation by the bowel is best obtained by the use of rather concentrated solutions of sodium sulphate. The water used for the solution of this drug must of course, be counted in the amount of fluid intake. The drugs of the digitalis group, especially apocyanin, may be useful adjuncts to the common hydrogogue cathartics. It is important that the volume of the excretion of urine and stool should be measured and contrasted with the fluid intake. To be sure, during infancy these measurements can only attain approximation, but they are to some degree a guide.

The applications of hot air baths or of hot packs to the entire body has two advantages; by sudorific action they help to relieve the kidneys of some of their duty of excretion of water and if they are carried out with care, these procedures are soothing to the irritated nervous system of the child. One of the useful ways of giving a dry, hot pack is to utilize the electric blanket. Properly applied, such a blanket produces a perfectly controllable heat. In the beginning a low degree of temperature is developed and little by little, this is increased. The body is thus inured to heightened temperature. Such a blanket provides a most effective and comfortable means for giving a patient, whose naked body should be swathed in a dry light blanket, a sweat bath.

Failing this apparatus, the ordinary hot water bag and hot bricks may be used or better than these, hot air delivered from a stove may be utilized. In the former instance, the clothing of the infant should be removed and it should then be enfolded in a woolen blanket and laid upon the bed. Hot water bottles or hot bricks wrapped in paper and cloths placed around the blanket, and the whole covered by another blanket will soon produce

perspiration. To give hot air baths safely, special apparatus is essential (see Methods). The use of the mustard pack is also a very useful alternative measure under these circumstances.

The convulsions sometimes encountered as a complication in cases of more profound kidney damage are favorably influenced by the hot air bath. Confronted by irregular twitchings, the premonitory signs of such a disturbance of the central nervous system, recourse should be had to lumbar puncture, a measure that frequently is successful in averting the onset of the convulsions as well as in relieving the accompanying headache. To reinforce the sedative action of the hot baths and the lumbar puncture, single injections of morphin are most useful (1/30 gr. for a 25-pound child). In the treatment of an acute nephritis, diuretics with specific effect on the urinary epithelium such as caffeine and theobromine, too frequently resorted to, are not only useless but do harm.

Among the commonest morbid manifestations in infants, **pyuria** holds a prominent place. No medical aphorism is better justified than that of Ashby, enunciated 25 years ago, that in the presence of an unexplained fever in an infant, **pyelocystitis** or **pyelitis** should be suspected. For this condition, together with acutely infected throats and inflamed middle ears, provides a great proportion of the cases of fever that occur during infancy.

Pyelocystitis is more prone to occur in girl babies than it is in boys, although boys are affected with greater frequency than is sometimes thought. While pyuria, revealing this condition, may be present at any age from one month throughout life, among infants it is more common between the tenth and twentieth months.

Opinions differ as to the portal of entry for infection. Whether or not the short, unguarded urethra of the girl gives direct access to infected matter, or whether in all cases the infecting agents are borne by the blood stream and localized later in the genitourinary tract, has been argued pro and con with some vehemence. There is plenty of evidence to show that infection may take place in either way. As the infective agent is most often the colon bacillus and as the bladder alone is affected in the early stages of the disease in a large proportion of female patients, and as physical conditions favor contamination of the interior of the bladder, there can be no doubt that minute amounts of feces may pass through the female urethra. Not always, however, does such passage into the bladder result in a cystitis. There are other factors involved.

The practical points are that the affected children are usually girls, most of whom on examination, will be found to be wearing outgrown diapers; these tend to drag the vulva open and to contaminate it. Examination of the urethra will reveal that the external meatus at this age lies deep in a little pit, most favorably placed to receive liquid feces or contaminated bath water. These conditions lead to the conclusion that practical preventive measures call for the avoidance of out-grown, too tight or improperly applied diapers and for the thorough cleansing of the soiled area before bathing; nor should liquid feces be allowed to remain in contact with the vulva for any length of time. Cleansing of the napkin area, too, should be in a direction away from the vulva. It must not be forgotten that a vulvovaginitis may be the initiating cause of a cystitis which may persist after the cause of the bladder infection has disappeared. A balanitis may act in a like manner.

It has been stated that if any large proportion of cases of cystitis followed direct contamination there could be no pyelitis as a complication because it would be impossible for the infection to ascend the long ureters to reach the pelvis of the kidney. Those who argue in this way have forgotten the work done by Bond several years ago when he demonstrated that suspended particles of India ink placed in the bladder reach the pelvis of the kidney on ascending mucous currents which sweep over the epithelium of the ureters. He showed that this type of transportation is available not only in the ureters but also in the ducts of the pancreas and gall bladder as well. However, even were these currents not present, there is another method and one which, it seems, is often effective in the evolution of a simple cystitis into a pyelocystitis; through superficial ulcerative lesions about the trigone, lymph channels are opened which permit the entry of bacteria into the blood stream. There is a further proof that access to the kidney may be had directly through such lymph channels, many of which surround the ureters, and thus bacteria may find their way into the renal pelvis. Lindberg, in 1889, showed that in ascending infections that involve the kidney, the infarcts are intralobular, the apex of each passing into a renal pyramid while in cases where the infecting organism is borne to the kidney by the blood stream, the infarcts are interlobular.

Direct blood stream infection of the kidney with a subsequent descending invasion of the bladder with cystitis may arise in the course of a proctitis, with fissure in ano, colitis or enteritis, al-

though it is probable that many cases of pyelocystitis that have been considered secondary to other lesions were themselves, primary.

A third method of infection by contamination direct from colon to bladder is rare in children although it complicates many cases in adults especially among old men, and in women after confinement.

Other organisms besides the colon bacillus, especially streptococci and staphylococci, are sometimes responsible for the presence of pus in the urine. In such cases, the infection is most often blood-borne and arises from foci, most frequently in the upper respiratory tract, although furunculosis or impetigo or osteomyelitis or any infective focus elsewhere in the body may be the initial lesion. Blood-borne infections often lead to the production of multiple renal abscesses—the Bruer-Barnard type of surgical kidney, a condition which is usually remediable only through drainage; although where the small abscesses occur in close proximity to the pelvis and open into it, spontaneous cure may result.

The *clinical manifestations* of pyelocystitis are varied. In young infants a rigor or an attack of alarming cyanosis may herald the advent of the infection. The fever may rise sharply and the curve later become septic in character. Urination becomes frequent and is painful; often it is accompanied by crying because the urine is very acid and irritating to the urethra; it may trickle into the vulva in girl babies and a great deal of irritation and even some purulent vulvitis may be induced. In such cases seen in the first few days, examination of the flanks will often reveal an enlarged tender kidney to the palpating hand. Babies with this type of painful affection of the bladder and kidneys tend to draw up their legs and to assume a fixity of posture with one or both legs; this taken together with the pain that follows movement of the thighs, may confuse the observer and lead him to suspect the presence of a scorbutic periostitis involving the femurs.

In such cases, examinations of the urine collected as advised in the chapter on Methods, renders the diagnosis clear. However, the symptoms are not always so revealed.

By far the most valuable information obtainable is to be had by *cultural studies of the catheterized urine* and this information is especially important in aiding the observer to arrive at a valid prognosis. It has been demonstrated that the colon bacillus and related organisms invade the kidney. Most often, they attack

the pelvic portions of the organ and rarely become the cause of abscess formation. Therefore, if culture demonstrates the presence of B. coli in pure culture, there is little need to fear the possibility of *surgical kidney*. On the other hand, if pure cultures of cocci are isolated from the urine in the presence of symptoms that denote a fulminant infection, there is reason to fear that multiple abscesses may develop; for when these bacteria find their way into the renal tissue, they are apt to localize at the glomerulus or near it in the more minute capillaries where they are prone to run on to suppuration.

Especially in older children, one finds *subacute* or *chronic* cases in which pus cells are scanty but in which the colon bacilli are abundant in the recently catheterized specimen of urine. In such cases the infection may last for years and give no suggestive symptoms, and some of the enureses that occur in older children are of this origin. This condition does not call for extensive comment as it is rare during the first 2 years of life. However, in small babies, subacute or chronic cystitis may present few symptoms that fix the observer's attention on the genitourinary tract. Careful investigation of the history will usually disclose that at some past period, symptoms analogous to those just described as revealing an acute pyelocystitis were present, but usually such a history will fail because many parents are notoriously unobservant.

In such instances, the physician may be consulted because of gastrointestinal disturbance, anorexia, vomiting, moderately frequent and greenish-appearing stools which give way after a while to constipation. Pallor, failure to gain, paroxysmal crying and a persistent fever are always present in this sort of infection and an examination of the urine will reveal pus and bacteria sufficient to sustain the diagnosis. The blood picture is that of a leucocytosis. Certain cases in this category present an intense intoxication which may readily confuse the observer and lead him to think of a typhoid fever, a condition which can usually be differentiated by the leucopenia, characteristic of that disease. But it must not be forgotten that in typhoid fever, a complicating cystitis caused by the presence of typhoid bacilli in the bladder may occur. It is very easy on simple microscopic examination to mistake the typhoid bacillus for the colon bacillus. It has been stated that in dilutions of 1 to 50 that the serum of a chronic colon cystitis case will agglutinate cultures of typhoid bacilli but that in dilutions of 1 to 100 this clumping does not take place, a

point that is worth consideration in arriving at a differential diagnosis.

Acutely oncoming infections of the genitourinary tract may present the clinical picture first described by Dupré as meningism and for 48 hours may be so confusing that only by lumbar puncture can a differentiation be made. This, however, is a condition hardly to be expected, although simple convulsions may be brought about in spasmophilic babies suffering with this as well as any other acute infection accompanied by toxemia and high fever. Occasionally, intractable vomiting recurrent in type, may be the revealing symptom of a chronic or subacute colon cystitis.

The methods of *treatment* have been subject to as much difference of opinion as have the avenues of infection and this is probably due to the fact that the common infective agent, the colon bacillus, probably occurs in many different strains and of different virulency. For the most part, if the cases are seen early, the use of citrate of potash, given in large quantities of water, will be successful in ameliorating the symptoms, although the colon bacillus may persist for years in the urine and defy every effort to eliminate it. The successful use of the citrate depends upon our ability to accomplish a prompt and thorough alkalinization of the urine with the drug. Still advises that the drug, well diluted in water, be given in 10 to 15 grain doses, according to the age of the child, every 2 hours day and night until the urine is thoroughly alkaline, a state of affairs that can be accomplished within 48 hours in most cases. The alkalinization of the urine can be hastened by the addition of bicarbonate of soda to the food or by the instillation of 1 or 2 ounces of a 5 per cent watery solution of the same drug into the rectum every 2 hours. The amount of alkali appropriate varies with the individual case. There is some tendency for disturbance of digestion to follow too intensive use of these drugs. A simple check is to be had by testing the urine with ordinary litmus paper. The attendant is provided with strips of the test paper and is instructed that as soon as the child passes a urine which turns the red paper blue, the dose is to be halved and the interval between doses doubled. However, even with the most thorough and prompt alkalinization of the urine there will be a certain proportion of the cases in which the symptoms will persist. For such, the use of hexamethylenamine (urotropin) is often curative. But it must never be forgotten that this drug may be damaging

to the glomeruli and cause hematuria. It has been demonstrated that hexamethylenamine is inefficient except when the urine is of high acidity; the drug should be given well diluted and in large dosage. It is our custom when using this remedy, to order 40 grains for a 10-month old child or 60 grains for a 20-month-old child and to have it dissolved in a pint of water which may be used as the diluent of the ordinary formula or be given to drink in place of plain water during the day. The inefficiency of hexamethylenamine in the presence of an alkaline urine makes it imperative that the patient should not be given the citrates or bicarbonates together with the urotropin. In fact it is often of value to produce an increase in the acidity of the urine by the use of acid sodium phosphate in doses of 2 to 3 grains every 2 hours. This drug, however, has a disagreeable taste and is apt to disturb the digestion. An alternation of these two methods of treatment is sometimes effective in abating symptoms and more especially in eradicating bacteria when they persist in the urine.

Occasionally neither citrate of potassium nor hexamethylenamine seems to have the slightest effect upon the symptoms and course of the disease. This is especially true when the cystitis is complicated by pyelonephritis or has become chronic without previous treatment or where treatment has been attempted with insufficient dosage of these drugs. Under such circumstances, the use of a powder composed of salol 3 grains; benzoate of soda, 5 grains, (for a 25-pound baby) repeated at 3 or 4 hour intervals during a week or ten days will sometimes be followed by most gratifying results. Under some circumstances the colon bacillus is so persistent that no method of drug treatment is entirely efficient in dealing with it. Under these conditions, a resort to the use of vaccines is indicated, but it seems useless to inject stock vaccines. Autogenous vaccines in ascending and large doses, the size to be determined in each individual case, should be tried and often they will achieve results when least expected; but on the other hand, these bacterial products are as frequently ineffective as they are successful.

When the infecting agent is an organism other than the colon bacillus, the disease is usually self limited. Ordinarily, the methods of treatment outlined for use against colon bacillus infections may be employed with confidence. The only exceptions are supplied where both kidneys are found to be infected and subject to **multiple renal abscesses**. The picture of this complication reveals

itself by a sudden onset with high temperature and a rigor accompanied by depression and malaise. The child presents an alarming aspect. There will be some days of fever and prostration, often delirium. The symptoms may remain inexplicable for several days although the demonstrable increase in the leucocyte count may suggest a pus focus somewhere else in the body. For a few days the urine will show no more pus than has been constantly present since the onset of the sickness; then suddenly the number of white blood cells in the urine will be tremendously increased. Sometimes for 1 or 2 evacuations, it may seem that little else but pus is being passed from the bladder. Quite rapidly the urine becomes less purulent, the fever falls and the other clinical symptoms subside and the patient returns to comparative comfort. Such a state of affairs may recur again and again until the child either attains an immunity and a cure is accomplished or it drifts into a general sepsis and dies. A similar alarming group of symptoms may occur without the presence of the revealing evacuations of pus in the urine. In such an instance, surgical kidney must be expected and the advice of a competent urologist taken; for when the abscesses lie deep and do not evacuate themselves into the renal pelvis, there is little hope for the child except through surgical intervention.

As an adjunct to drug treatment in many cases of cystitis, bladder washing is an essential procedure. Used once or twice in the beginning of treatment with possibly an occasional repetition, it is often decisive in bringing about a cure. For technic of bladder lavage, see Methods.

Some instances of persistent pyelitis in which it is possible to exclude the presence of kidney abscess, show no improvement until the kidney pelvis is subjected to lavage. This is a procedure that should be undertaken only by an expert urologist but in such hands, the writers have observed it to be a measure of great value.

The appearance of blood in the urine may vary from a slight smokiness due to the presence of a moderate number of red blood cells to the bright red that follows a copious hemorrhage. During the first two years of life, **hematuria** occurs chiefly as a complication of acute glomerular nephritis. There is a well recognized form of chronic glomerular nephritis which is accompanied by a mild but persistent hematuria without other symptoms. This disorder is definitely the result of congenital syphilis. It rarely appears before the end of the second year, but may make itself apparent at any time. Drugs, especially hexamethylenamine,

which are given in order to combat a cystitis, may be the cause of severe hematuria. It is a question whether this bleeding is not from the lower urinary tract. Urotropin is irritating only when changed into formalin, and very little of it is so altered at the kidney level. This also explains its ineffectiveness in case of pyelonephritis or pyelitis and its value in simple cystitis. Almost any sample of urine, carefully searched, will reveal a few red blood cells and if search be made of the urine of a child who has an acute inflammation of the upper respiratory tract, or in the presence of a pus focus elsewhere, the occasional presence of even a considerable number of cells need not surprise the examiner.

Still has reported hematuria following paroxysms of pertussal cough. The various flukes of the Orient and Africa may sometimes be found in a hematuric urine. The Bilharzia is a common source of bloody urine in children who live in Africa and is seen among the Asiatics. It has been found in the urine of Asiatic children on the Pacific coast.

A very common source of slight hematuria arises from *trauma of the urinary meatus* by friction of the penis on a tight diaper, especially in the circumcised. If the excoriations are deep enough, a fissure, from which drops of blood are exuded and passed with every urination may be produced.

In the newborn hematuria may be a part of the clinical revelation of a *hemorrhagic disease.* In older infants, it is one of the symptoms of *scurvy.* On rare occasions, the passage of a **urinary concretion** or of crystals of uric acid or of oxalates may traumatize the urinary way, an accident that will be followed by the appearance of more or less blood in the urine. Rare even in this unusual group, is the appearance of calculi formed by the aggregation of ammonium urate crystals or by the deposit of phosphates about an organic core, the result of infection. The diagnosis of urinary calculi in infancy is a matter of extreme difficulty especially during the first year of life and fortunately one that the physician is almost never called upon to make. There is on record at the University of California Hospital a case history of a baby of 8 months who spontaneously passed three large stones. Three months later nephrectomy and ureterotomy were done and at this time three more calculi were dislodged.

The pain of the *ureteral colic* will usually be mistaken for intestinal colic although the hematuria in such a condition may

lead to a suspicion of the true state of affairs. Ureteral obstructions are apt to occur and hydronephrosis to develop. Intermittent obstructions to the outlet of the bladder have been reported, a symptom suggestive of which is painful erection in males on attempts to empty the bladder. There is extrusion of milky phosphate-bearing urine completing micturition. Radiographic aid will be of determining value in the diagnosis of a suspicious case. The treatment is essentially in the hands of the surgeon.

As hematuria is but a symptom, the *treatment* of the condition refers to the cause. Acute glomerular nephritis is best treated by the limitation of fluid and the preservation of a proper diet and by encouraging the skin and bowel to eliminate water. Chronic glomerular nephritis often succumbs to antiluetic measures. The excretion of crystals may be minimized by increasing the dilution of the urine and by the use of such drugs as cinchophen (atophan) when the crystals are of uric acid and by the use of phosphoric acid when the crystals are oxalates. Cinchophen should be used in 1 or 2 grain doses 4 times a day and a dilute phosphoric acid, in doses of 1 to 3 drops well diluted in water 4 times a day.

In the treatment of hematuria as a part of a hemorrhagic disease no other remedy is nearly so effective as the intramuscular injection of whole blood. For details of this procedure, see chapter on Methods. This is a therapeutic measure without danger and entirely superior in effectiveness to the injection of horse serum or sterilized gelatine.

When the hematuria is part of a scurvy, the vitamine-bearing foods which are given to remedy the hemorrhages elsewhere, will effectively abate this particular symptom.

Tuberculosis of the kidney as a source of hematuria during the first 2 years is almost unknown except as a part of the picture of a generalized tuberculosis. Together with a pyuria, it may appear as a terminal event in the course of such an infection.

Tumors of the kidney are relatively uncommon during the first 2 years of life. Ordinarily, the first symptom that confronts the physician is the presence of an abdominal tumor. Frequently this tumor has become very large before it is brought to the attention of the medical man. Pain is rarely a feature. Urinary changes are conspicuous by their absence, although profuse hematuria may occur as an early sign, especially in the sarcomata. Emaciation, constipation and vomiting are among the symptoms that develop during the later stages, and at this time the absence

of pain may give place to much abdominal distress because of the pressure of the rapidly growing tumor on the abdominal viscera.

The recognition of the renal origin of the tumor is rarely difficult. Some part of the mass always will be palpable in the flank and the costo-vertebral angle will be fuller than normal when examined by the palpating hand. The colon will lie in front of the tumor, and its relation to the mass can be demonstrated by inflation or by the use of enema and radiogram. This relationship differentiates an enlarged spleen. It is important that a thorough urologic study be made when a renal tumor is suspected.

The tumor itself moves little with respirations. Suprarenal tumors are less apt to fill the flank and are palpable more toward the middle line. Tuberculous peritonitis, with large masses of glands and many adhesions may prove puzzling, but here again the palpation of the flank and the relationship of the colon will aid the examiner in making a decision. On the right side, tumors of the liver are very difficult of differentiation. They are supposed to move more freely on respiration than do the kidney tumors, but as a matter of fact, the liver tumors may be a metastatic complication of the kidney tumor. The refinements of differential diagnosis are not of great practical importance. The presence of a tumor in the kidney region is warrant for surgical intervention if the diagnosis is made early enough but if the child is brought to the physician only after the mass has attained considerable size, it will be certain that metastases have already taken place. The authors believe it is humane and good medical practice to state the facts clearly to the parents and to advise against operation which can do no more than prolong the pain of the child.

The pathologic variations in these tumors are of great interest. There is much in the literature relating to them. Unfortunately there is nothing that aids much in determining considerations of treatment.

One very interesting neoplasm, the signs of which are often mistaken for the signs of kidney tumor because of their similarity, is **new growth in the adrenal gland.** There are two very distinct types of tumor, one involving the cortex of the gland and the other the medulla. The latter is rather frequent in young children. These tumors were clinically classified by Robert Hutchinson in 1907 and their interest lies in the manner and influence of the metastases. Unfortunately, these metastases often occur

before the principal growth has given sufficient symptoms to permit diagnosis.

The earliest revelations of the disease may appear as pains, most often in the legs. Sometimes these pains do not occur however, and proptosis, usually unilateral, is the sign that first arrests attention. Preceding this protrusion of the eye or occasionally together with it, there may be some hemorrhage about the lids. Hutchinson stated that the eye first affected is on the side of the primary suprarenal growth. Frew confirms this and explained it by the difference of lymphatic drainage of the two sides. Very soon the bones of the skull, especially about the temporal fossae, give evidence of metastatic growths. Even at this stage of the disease, the palpating hand over the kidney region may discover no evidence of a tumor. Unlike Addison's disease, pigmentation is not a feature, neither are there any changes in the blood pressure, nor is there leucocytosis or marked fever, although there may be some slight increase in temperature. The further progress of the disease is marked by increasing exophthalmus which often prevents closure of the lids and protection of the eye, a state of affairs which may lead to corneal ulceration. Optic neuritis is a common development and proceeds to such a degree that blindness may ensue. Death is never delayed for more than a few months after the first signs of the disease have become evident. Hutchinson further observed that when the right suprarenal gland is involved, the metastatic processes are less apt to dominate the clinical picture and the suprarenal tumor is more readily palpated at a relatively earlier stage in the course of the disease. It is interesting that a group of enlarged glands appears behind the sternal end of the right clavicle when the right adrenal is the site of the tumor. At their onset, the eye signs may be mistaken for the exophthalmus of scurvy, but the absence of the general tenderness of scurvy and the rapid development of metastases that accompany these adrenal tumors, will make differentiation a simple matter. Because of the similarity of the orbital and skull symptoms, chloroma may be mistaken, but a study of the blood will prevent confusion.

The **tumors of the cortex of the adrenal** are of especial interest for two reasons; first, because their pathologic nature is obscure and they are difficult of classification; second, because the tumors whether malign or benign, interfere with the function of the gland and produce a series of secondary changes affecting growth and metabolism. As a result, certain of these children develop

precocious puberty; others secondary sex characteristics without a true premature sexual development; some become obese; and others show excessive muscular development without obesity. True sexual prematurity is a function of the overmuscular type of case, while in the obese, sex characteristics fail. The most usual and striking feature, however, is the obesity which is different from the simple fatness of the ordinary fat child; the adipose tissue is distributed as it is in the obesity of middle-aged adults. Occasionally identical changes occur in tumors that originate in other glands of internal secretion, especially the testes, ovary and pineal gland.

The results of **hemorrhage into the suprarenal gland** have been treated in the chapter on Hemorrhages. Still suggests a useful clinical classification; first, cases in which the affected die within a few hours after birth (possibly with hemorrhagic disease); second, cases that are secondary to an infective process elsewhere; third, cases that run an acute pyrexia for a few days, often with purpuric skin eruptions and in which suprarenal hemorrhages are found postmortem.

The symptoms of the acute infective type consist of a pyrexia with vomiting and occasional purplish mottling of the skin. Convulsions are the rule, and collapse with weak and rapid pulse supervenes running to a fatal termination usually within the first 24 hours. The treatment is purely symptomatic.

Developmental defects of the genitourinary tract are not common. There may be interference with the development of the ventral wall of the urogenital canal with a resultant failure of closure at the median line fissure and an absence of the anterior wall of the bladder and the abdominal wall covering it **(exstrophy of the bladder)**. There may also be a defect in the symphysis pubis; the resulting opening may be extensive, involving the whole length of the urethra with a complicating **epispadias**. The penis is short and deformed, the prostate ill-developed and the prepuce defective. Sometimes the scrotum may be absent and the testes only partially descended. When the child is a female, the urethra is cleft and forms a continuation with the lower part of the exposed bladder wall. The sphincter vesicæ is absent, the ureters open into the exposed surface and the urine flows away readily. These facts render the recognition of the condition inevitable. Obstruction to the outlet of the ureter occurs and there is a constant danger of the production of a secondary hydronephrosis. Prolapsus of the bladder happens rarely and is of no

practical importance except in those cases in which there is a distended ureter and a hydronephrotic tumor which forces an extrusion of the bladder through the urethra. Epispadias, when it occurs, is usually a part of an extroversion and hardly ever seen otherwise.

The *treatment* is entirely surgical. Plastic reconstruction of the bladder is of no avail as the sphincter is lacking, and the operation of choice among urologists is now the transplantation of the ureters into the rectum. The risk of an ascending infection seems to be less than would be anticipated from a knowledge of the bacterial condition of the rectum.

Congenital strictures of the male urethra are not unknown. The site most often involved is at or near the meatus; when they occur here, they are readily overcome by simple incision.

Hypospadias is seen with moderate frequency. There are several varieties. For purposes of surgical treatment, according to the part of the urethra most involved, hypospadias has been divided into perineal, perineoscrotal, penile, penoscrotal, and glandular. In the latter, the glans is fissured on the ventral surface and spread out, and the urethra terminates at its base in a pin point opening which may have to be enlarged in the first few days of life. In such a case, the prepuce and frenum may be absent. Occasionally, a blind pouch terminates at the normal meatus, while the true opening of the urethra lies posteriorly.

In the more unusual forms, perineoscrotal and penoscrotal, the urethra ends in the perineum or at the base of the penis. In the true perineal type, the condition may be mistakenly considered "hermaphrodism," for the scrotum is fissured and the superficial appearance of the genitals is that of a vulva.

Surgical *treatment* offers the only aid. It is of some importance to determine the age at which the patient should be subjected to surgical interference. It is the writers' opinion that slight obstruction should be relieved by simple urethrotomies and attempts at plastic reconstruction should be delayed until about puberty because of the delicacy and incompleteness of the tissues and because of the deformities that may follow growth.

Both testicles should be in the scrotum at the time of birth and should remain there. In certain children, the cremaster muscle is active with the result that the testes move freely from the scrotum to the canal and back again. Such a testicle must not be considered undescended. A true **undescended testicle** cannot be drawn down into the scrotum during early infancy. Its

misplacement is the result of some interference with its descent so that it may remain at a point along its normal course in the abdomen, the inguinal canal or the upper part of the scrotum. On the other hand, it may be diverted from its normal path and be found in the perineum.

Truly undescended testes are so ill-developed that they are functionless and surgeons regard them as possible sites for sarcomatous degeneration. They are often found complicating hernias and are attributed an influence in producing ruptures. The descent of an ectopic testicle may take place normally during the growing period and for this reason it is wise to delay operation for their replacement until the 5th or 6th year.

Hydrocele of the cord is not at all infrequent among young children. The hydrocele itself is an accumulation of fluid in some part of the tunica vaginalis. It is convenient to consider the hydrocele as one of three types; first, the congenital form in which free communication is maintained between the peritoneal cavity and the fluid accumulation around the cord; second, the infantile form in which such a communication is lacking; and third, hydrocele of the cord in which there is an obliteration of the lower part of the sac of the tunica vaginalis and in which, therefore, the testicle is apart from the swelling produced by the fluid. This last condition results in a cystic tumor lying in the canal; it is rather limited in movement and like all forms of hydrocele, it is translucent. In the congenital form, hernia is not an infrequent complication. Spontaneous disappearance is the rule in hydrocele and therefore it is best to omit any surgical procedures for radical cures at least during the first 2 years of life. If the fluid accumulation persists into later childhood, aspiration of the fluid provides an effective cure which is applicable especially in hydrocele of the cord.

A **redundant prepuce** is a common enough physical fact. The mere presence of a lengthy foreskin does not warrant its amputation. Whether overlong or not, the prepuce may be contracted and cause **phimosis**. During early infancy, however, if the skin over the glans is sufficiently retractable to allow the passage of the urine, the case should not be considered one of phimosis. Very often, narrowing is so great that it causes difficult and painful urination. Sometimes urinary concretions may form because urine is forced between the glans and prepuce and phosphates left in gritty masses to accumulate around the coronal sulcus.

Simple dilatation and retraction is all that is necessary in most

cases in order to make it possible to properly cleanse the glans and remove smegma from the base of the corona. When there is a great deal of narrowing and many adhesions, dilatation and retraction are painful and it is well under these circumstances to circumcise; but the writers have no sympathy with the general circumcision of young children, and protest against the all too popular idea that irritation of the glans in phimosis is the cause of the host of nervous and reflex disorders for which circumcision is so often recommended and practiced. **Balanitis**, that results from uncleanliness is no excuse for unconsidered circumcision. Cleanliness and the use of a simple phenol ointment will overcome the inflammation and the superficial ulceration of the glans penis. As a preventive measure, the obstetrician should make it his business to see that the baby's foreskin is well retracted and that the nurse or mother is shown how to draw the foreskin back in order to expose the full extent of the glans. They should be instructed how to clean it and how to return the prepuce properly. If this instruction were routinely given, there would be little balanitis and little need for treatment by circumcision.

Occasionally a heavy **meatal opening** so interferes with free urinary flow that micturition becomes a painful act and under these circumstances, meatotomy must be practiced.

Paraphimosis following unskillful retraction is fairly common among boy babies. It is painful, but it usually rights itself. Cold compresses tend to reduce the edema and when the swelling is very great, a finger bandage soaked in iced lead and opium solution wound on and off the penis several times will force the edema out of the organ and permit a ready replacement. If the paraphimosis occurs and recurs, circumcision should be advised.

Acute orchitis, in the first 2 years of life, is practically unknown even as a complication of mumps. Occasionally, a tuberculosis of the peritoneum advancing along the spermatic cord involves the epididymis but this, as well as gonad inflammation in congenital syphilis, is a medical curiosity.

The **vulvovaginitis** of little girls provides one of the most perplexing and discouraging problems with which the medical man has to deal. This is especially true in cases in which Gram-negative, intracellular organisms are demonstrated, more especially if these organisms have originated in a case of known gonorrhea. There is some doubt whether all Gram-negative, intracellular diplococci seen in smears are truly examples of Neisser's coccus, for certainly many times microscopic evidence of such

organisms can be had in cases where the clinical signs of gonorrheal vaginitis are entirely lacking except for the presence of a small amount of pus. In these cases, there has never been anything comparable to the acute stage of vaginitis that is seen arising in children whose infection is known to have been derived from an undoubted case of adult gonorrhea. Furthermore, such doubtful cases are often discovered in families or in institutions in which little care is taken to prevent the spread of infection, and yet no house epidemic follows their presence. This is a phase of the subject that remains to be carefully worked out by experimental methods before any definite pronouncement can be made.

The symptoms of **gonorrheal vaginitis** in little girls during the stage of onset differs very little from the picture seen in adults. It begins with fever, acute irritation of the vagina and vulva and the presence of pus. The microscopic picture is unmistakable. Without the microscope, however, a gonorrheal vaginitis must not be diagnosed as very considerable vaginal discharges may follow contamination of the vulva by colon bacilli and by the ordinary pus-producers. Local irritation caused by oxyurides is also a fertile source of pus production in this region.

Credé prophylaxis of the newborn, so effectively applied to the eyes, should also be extended to include cleansing and silver applications to the vulvas of newborn girls, especially to every case in which there is reason to suspect a gonorrheal contamination of the mother.

The *treatment* of vulvovaginitis is most unsatisfactory. Tampons, irrigation and instillation are all recommended, none, however, with any great enthusiasm. In a woman infected in this way, it is possible to treat the os uteri by local applications, but in infant girls, such a procedure is almost impossible, and were it possible, hardly advisable, because of the minuteness of the orifice and the readiness with which an infection may be forced into the relatively wide Fallopian tubes; and yet, the persistence of the infection is explainable in all probability by the fact that mucous glands of the os offer a comfortable and permanent home to the coccus of Neisser.

The writers have found irrigation the treatment most effective in this disease, but this irrigation must be carried out with meticulous care. After a preliminary cleansing of the vulva, a female urethral speculum of appropriate size is inserted. (See Methods.) Through this is passed a fine male catheter which is allowed to reach the vault of the vagina. The speculum is then

withdrawn to a point where the hymen is just kept open. A pint of irrigation solution is then allowed to flow through the catheter from a douche tin or Kelly bottle kept at a pressure head of not more than from 9 to 12 inches. Free and constant egress of the fluid from the vagina must be insured, otherwise, infected fluid may be forced into the uterus and into the Fallopian tubes. Following the irrigation a small strip of gauze soaked in 20 per cent argyrol or 1 per cent protargol should then be passed with a fine forceps through the speculum as it is withdrawn. In this way, the walls of the vagina are kept from contact, and the medication is applied to all the vaginal surfaces. For irrigating, the writers prefer normal saline solution, but there is no objection to the use of potassium permanganate or to a dilute solution of organic silver. In persistent cases the use of a gonorrheal vaccine may have to be considered. Experience leads us to believe they may have some value in certain chronic cases.

Recently a treatment has been employed with increasing success in the juvenile venereal ward of the Cook County Hospital. It consists of the instillation of silver nitrate, 1 grain to the ounce of vaseline. The ointment is injected by aid of a common glass syringe, to the tip of which is attached the end of a soft rubber catheter. The syringe is filled by means of a wooden tongue blade. The vagina is injected with the ointment. Such treatment is continued for about a week, when the silver nitrate is replaced with a 1 per cent mercurochrome.

Tuberculosis of the **penis** is an affection that occurs occasionally, particularly in the ghetto districts. It is usually the result of ritualistic circumcision. The tubercle bacillus is carried into the wound with the saliva of an officiating, phthisical rabbi. The glans, corpora cavernosa and inguinal structures soon become involved. There is marked induration and sloughing of glandular tissue.

CHAPTER XIX

DISEASES OF THE OSSEOUS SYSTEM

The characteristic conformation of the infant's body is largely due to peculiarities of the infant skeleton. These peculiarities are the result of natural provisions for growth in the osseous system. At birth the epiphyses of the long bones are cartilaginous structures whose function is to produce increase in length; they are quite as important as any other element of the skeleton. Injuries or infections of these structures may give rise to deformities because of the inhibition of growth of the length of the bones. The flat bones, especially those of the vault of the skull, which originate in and develop from membranes rather than from cartilage, are also subject to interferences with their normal development. The sutures and fontanels which are spaces placed so as to permit a sufficient growth of the skull may close prematurely or their closure may be delayed until late infancy or early childhood. The growth of the skull bones takes place from osseous centers which lie well away from the periphery of the bone.

Bossing of the skull is a condition in which much proliferation and thickening occur about these centers of ossification, and this is accompanied by slow and inefficient osseous development in the outlying portions of the bones of the vertex. This condition is an almost constant accompaniment of rickets, and it may be seen in certain premature and weakly infants as early as the third or fourth month of life. More often the development of the deformity is delayed until after the seventh or eighth month. Bossing is almost invariably accompanied by delay in the closure of the fontanels and the cranial sutures. This combination it is that gives the characteristic appearance of the head in rickets—the full frontal and parietal region and the flat area on top. Extreme degrees of bossing produce the type of skull known as hot-cross-bun head. Parrot, who originally described this deformity, thought rickets to be the result of syphilis and this type of bossing to be evidence of the latter disease, a view no longer considered tenable. Bossing even in scorbutic or syphilitic patients is most often an evidence of rickets, although achondroplasics and individuals with cleidocranial dysostoses may show a similar thickening about the ossifying centers of the cranial bones.

Early closures of the fontanels and premature union of the sutures can occur as a part of the clinical picture of a **microcrania**. Usually such a small head occurs in a patient with congenital hypoplasia of the brain and a microcephaly.

Localized **faults in closure** of the interfrontal or interoccipital **sutures** may occur, leaving undefended spaces which permit **meningocele** to develop.

Imperfections of ossification occur in the bones of the vertex with the result that in places thin areas appear. These may be so marked that they dent under finger pressure to spring out again when pressure is released. The sensation is like that felt when a thin sheet of parchment is pressed upon. The most usual site for **craniotabes** is about the lateral fontanels involving both parietal and occipital bones, although either bone may be affected without change in the other. If the condition occurs early, it may be a complication of congenital syphilis; if it appears later, it may be present in rachitic patients. It is probable that neither disease is specifically responsible for the manifestation. Any morbid process that influences the power of the osteoblasts to produce bone in the primitive membrane may be influential in producing craniotabes.

The metastases of malignant *adrenal tumors* (*neurocytomas*) appear in the skull bones, especially about the frontal region near the orbit. Such metastatic deposits are usually the revealing signs of the disease, as the increase in size of the primary adrenal tumor is so insidious that it may easily escape observation in the stages antecedent to metastasis.

Certain children come into the world with **fragilitas ossium**. They possess bones so fragile that they are unable to meet the essential strains to which life subjects them. Such bones fracture under the slightest stress, some even in intrauterine life so that an infant may be stillborn or appear at term with many fractures. In some cases even more than 100 breaks have been counted. The ribs seem especially prone to this fragility although the long bones also are invariably affected. The underlying defect is referable to some congenital imperfection of the perichondrium which fails to take on the function of the periosteum at the proper time. The fractures occur near the epiphyses or in the bony shafts and may be complete or green stick. Once the bones break, they unite rapidly but the production of deformity is the usual result of union, either from malposition or from the produc-

tion of thick rings of calcified fibrous tissue which do not go on to perfect organization as bone. Another factor in the production of the deformities is the softness of the bones, which is sufficient to permit bending. The nature of the underlying dystrophy remains a mystery inaccessible to treatment. When multiple fractures are present at birth or come on in very early life, the prognosis is bad, but when the first breaks appear only after the child is some months of age, the outlook is that the infant will overcome the condition and acquire a normal sturdiness of skeletal structure. The *treatment* is the surgical procedure appropriate for the fracture or fractures that appear in the particular case under observation. Rickets, syphilis and scurvy may produce bony changes that lead to multiple fracture, but there is little chance that any of these will be mistaken for fragilitas ossium.

Single and multiple fractures in childhood also result from the ravages of simple cysts of the bone.

Closely related to the condition that leads to multiple fracture is the **osteomalacia of infancy.** This morbid entity is also known as **mollities ossium.** The evidences of this disease rarely appear before the child begins to walk, and for the most part the onset is delayed until childhood. The cause is a mystery; some have sought to relate it to altered ovarian secretion, although the infantile cases occur as often in males as females. Similar softening has been described in the wake of the infectious diseases. The softening is a striking feature of the disease, for the bones may be as easily cut as an ordinary cheese. The periosteum is thick, while the bones are thin as well as soft and rarefied. In a young infant still maintained in the recumbent position, affected with this disease, the tibias may be excessively bowed by the pressure of one leg on the other; or when the infant is first put on its feet, the body weight may be too much for the soft femurs which bend easily. A little later, the weight of the trunk distorts the spine, and the pelvis is twisted out of shape. The clavicles, the humerus, the femur, in fact any or all of the bones may suffer. With the onset of these deformities, pain develops from the muscular stress and the imperfect skeletal support, and this lack of support together with the muscular weakness is such that fatigue readily supervenes. The same condition may appear in fragilitas ossium, but in that condition early fracture is always a feature, while break of the bone never appears in osteomalacia until after extensive bending has taken place. It is probable that

most if not all the cases which have been considered as tardy rickets are in reality instances of osteomalacia or of nearly related morbid states.

The *treatment* is naturally the assurance of measures best calculated to promote nutrition and to protect the infant from deformity; rest in bed and appropriate diet are essentials. Cod-liver oil and phosphorus, so valuable in the treatment of rickets, seem to have no influence on the processes of calcification in osteomalacia. Adrenalin injections have been recommended, but one of the writers, after using the drug in the treatment of osteomalacia, was unable to detect that it had the least influence over the disease.

The **teeth** are developed in the infantile jaws from certain primitive dental structures, the dental sacs. These sacs lie in irregular depressions distributed along the alveolar processes of the jaws. The sacs representing the future deciduous teeth lie superficially covered by connective tissue and the gingival mucous membrane. Deeper in the alveolar processes lie other sacs, the germs of the permanent teeth to be developed at a later period.

The time of the **eruption of the teeth** and the order of their appearance is a familial function, and while it is true that malnutrition and rickets do delay the appearance of teeth and in some instances interfere with the regular order of their eruption, this delay in interference is less frequent under these circumstances than is generally supposed. Many well-nourished children whose skeletons are perfectly normal do not bring forth their first set of teeth until quite late in infancy, while some of the undernourished and rachitic are possessed of their full quota very early in life. Malnutrition in the first months of life is more influential in delaying the appearance of the teeth than is malnutrition or disease that develops in the latter part of the first year. It is an arresting sight sometimes to see the fully erupted, beautifully developed teeth in the mouth of an atrophic, wasted infant suffering from some nutritional disorder.

In most instances, however, it may be accepted that a child should have 20 teeth erupted by the time it is 30 months of age, and that of these, 16 should have appeared by the end of the second year. Six of these teeth should have been through the gums by the time the infant is a year old, and 12 by the time it has reached its eighteenth month. Infants may be born with one or more teeth, but this is a very unusual manifestation. Some children, not all of them by any means otherwise precocious, de-

velop their 2 lower central incisors during the third or fourth month; but for the most part these 2 teeth make their appearance not earlier than the sixth month, while many healthy babies fail to show them before the ninth or tenth month. Within 4 or 5 months after the appearance of these first teeth, the 4 upper incisors should have erupted, the 2 central preceding the 2 lateral by 2 or 3 weeks. After this, the 2 lower lateral incisors should appear within 3 or 4 weeks. In the normal course of events these 8 incisors are all present by the thirteenth or fourteenth month, and they are followed, after the lapse of from 3 to 6 months, by the 4 canines—the "stomach and eye" teeth so dreaded by nurses and mothers. Another period of some months elapses before the 4 molar teeth appear to complete the eruption of the first set.

The dental sac undergoes continuous calcification until the tooth is erupted and is fully grown. Growth arises from the increase in length of the subgingival portions of the tooth. The increasing pressure of the outgrowing tooth causes absorption of submucous and mucous tissues along the alveolar margin and the tooth breaks through, very often without any local disturbance or discomfort to the child. Sometimes for a day or two before the eruption, there is tenderness and swelling of the gum margin, and a dryness of the mouth may follow after a preliminary stage of salivation. Except in those rare instances in which the tissues have been bruised and an accumulation of fluid or blood develops above the unerupted crown of the tooth, there is no excuse for incising the gums, and "lancing the teeth" should be considered a relic of medievalism.

To say that the eruption of teeth is never a cause of disturbance to infants is quite as unwarranted as it would be to accept the view that every rise in temperature, disturbance of digestion, and explosion of the central nervous system that occurs in infancy is directly due to the "reflex irritation" of erupting teeth. It is probable that nothing has been more disastrous to infant life than the propagation of this latter fallacy not only throughout the laity but also among many members of the medical profession.

The symptoms of disturbed health definitely referable to the eruption of teeth are for the most part entirely local, although slight degrees of fever with anorexia and a temporary halt in weight increase may accompany the local manifestations of dentition. Almost invariably there are a few nights of broken sleep; sometimes a continued wakefulness with intractable crying oc-

curs, but usually a few days of swollen gums, and salivation or dry mouth, with a disinclination to eat because of the discomfort, are all that is seen. If this disinclination to food is not respected and if feeding be urged, it may very well happen that gastritis and diarrhea will be brought about and a real illness develop. During the time the upper incisors are developing, it is no rarity for a nonfebrile coryza to take place, for the same nerves that supply the nasal membrane arborize to reach the mucous membrane of the upper jaw.

That dentition alone can cause *convulsions* is impossible, but no one of wide clinical experience can doubt that there are instances in which the slight added depression produced by the eruption of teeth acts to precipitate an attack in spasmophilic or rachitic children or in those with an inferior development of the nervous system. It has also been observed that some eczematous children show an exacerbation of the skin eruption with the appearance of every new tooth; however, this may be incidental.

The **teeth** themselves may erupt with **enamel insufficiently developed** and, as a result, early decay may set in. Occasionally, the teeth show decay coincidentally with eruption; such a condition demands great cleanliness of the mouth and the use of dental prophylaxis from the very beginning. A decision whether certain teeth should be extracted or left is a matter that demands good clinical judgment. The presence of the roots of the first set of teeth brings about conditions in the jaw advantageous for the proper development of the unerupted permanent teeth; therefore, wherever possible, the deciduous teeth should be maintained until they loosen spontaneously. On the other hand, the presence of numerous decaying roots, each bathed in pyorrheal pus, is distinctly deleterious not only to the health and nutrition of the child, but to the permanent teeth themselves. If these teeth come through into infected gums unprotected by mature dentine, they are almost certain to become the early victims of extensive decay.

There is a method in use among European dentists for the treatment of irreparable deciduous teeth which might well find a wider vogue in this country. The very earliest signs of decay are observed, slight excavations are performed, and repeated applications of 20 per cent to 30 per cent nitrate of silver are made. This procedure is often effectual in checking the further spread of the disease, with the result that the roots of the first set of teeth within the gums remain healthy enough to assert their normal function in relation to the second set. These are permitted a slow

development, inhibited from crowding, and insured against eruption into tissues alive with the germs of decay. Unfortunately the application of the nitrate of silver blackens the teeth, but the result is certainly not any more disagreeable to the eye than that which a widespread decay will inevitably produce.

Cleft palate is a developmental anomaly resulting from imperfect closure of the superior maxillary processes. It may be complete, in which case there is also a failure of union of the superior maxillary and frontonasal processes, either bilateral or unilateral. This is practically always accompanied by harelip, similarly either unilateral or bilateral. All gradations are seen, from a cleft which barely reaches through the velum palati to one which throws the buccal and nasal cavities into one and causes also a bilateral harelip. It is most usual to have a complete cleft palate with a unilateral harelip which for some unexplained reason is most commonly on the left side. The condition is distinctly hereditary, a history of a similar condition or of some other congenital anomaly being practically always elicitable. The lateral incisor teeth are missing as a rule, and the portion of the maxillary arch attached to the septum contains the median incisors. It occasionally happens that the buds for the lateral incisors are split and two small teeth appear. It has been interestingly observed that parents presenting a lack of lateral incisors sometimes produce offspring with harelip or cleft palate.

The optimum time for operative *treatment* is from the second to the twelfth week. Later than this, the case is always complicated by a varying degree of malnutrition which must first be combated before operation can be attempted. For these infants suckling is always difficult and frequently impossible so that gavage feedings are required at times. If the cleft in the palate is not complete, special nipples which carry on them a flat rubber diaphragm to close the palatal defect and permit of suction may be employed, often with success. The use of the Breck feeder may also be of advantage for very weak infants. As can be seen readily, it is advisable to operate early while the child is still in good nutritional condition. As a preliminary to operative procedure for the repair of cleft palate, it is sometimes useful to apply pressure by some form of elastic apparatus. The pressure appliance should not be worn continuously. It is best applied during periods of 5 to 10 minutes and adjusted so as to bring pres-

sure upon the superior maxillary processes. This method, repeated several times a day, will accomplish a surprising amount in reducing the width of the cleft. It is always advisable to have the repair of the palatal cleft performed before that of the lip. Should a small perforation remain after the operation, a closure can usually be brought about by means of the actual cautery. It has been determined that variations in the voice depend very largely upon the tension which is put upon the palatal velum. Since this has been recognized, operators are paying particular attention to this point, and comparatively normal speaking voices are now obtainable.

Congenital defect in the development of the clavicles is sometimes familial; it may be so extreme that the bone is entirely absent. Under these circumstances, abnormalities of growth in the membrane bones of the skull also take place and the condition is then known as **craniocleidal dysostosis**. Individuals so affected are possessed of extreme mobility of the shoulders, and physical examination reveals the clavicular defect. Inspection of the head shows a bulging bossed forehead with a central depression, prominent supraorbital ridges, and a relative exophthalmos. The condition is apt to be first noticed on an attempt to raise the infant by lifting it, and whoever makes this attempt with his hands in the baby's axillas will discover the undue mobility of the shoulder girdle. Once this is found, the unusual narrowing of the shoulders is striking. Nothing in the way of *treatment* offers the slightest chance of improving the patient's condition.

Congenital absence of the scapula with absence of parts of the trapezius and of the other muscles normally attached to that bone occurs. A child with this condition has been seen in the outpatient department of the San Francisco Children's Hospital. Absence of the scapula is a much rarer state than the deformity which gives rise to the clinical picture called **congenital elevation of the scapula**. The deformity is usually unilateral and the scapula is small, imperfect in shape and has a much higher level than normal. In some instances, a bridge of bone unites the vertebral edge of the scapula with the lower cervical and upper dorsal spines. Under these circumstances, power to elevate the arm is limited and a compensatory scoliosis occurs.

Children may be born with **absence** of any one or all of the **bones of a limb** or **limbs**, and the resulting deformity will be greater or less according to the amount of deprivation.

Certain children present the characteristic bony deformities of **achondroplasia.** These deformities are most evident in the femur, the humerus, and the bones of the base of the skull. The femur and the humerus both suffer in such a way that the growth in their long axis is interfered with. The result is a shortening of the limbs which appear out of due proportion to the length of the normally grown body. In infant patients who are usually fat, there is a peculiar distribution of the tissues over the shortened long bones, especially over the femurs. A number of circular furrows appear on the thighs and on the upper arms which look as though threads had been tied around the skin of the limb, constricting it. Between these furrows, the tissues bulge, and the normal fatty appearance of the thigh is thus exaggerated. When the infants are old enough to stand erect, it will be noted that the buttocks are prominent and present the same appearance of overlying fat. There is a marked lordosis of the lumbar spine and, as a result, an undue prominence of the belly. The lordosis results from a characteristic deformity of certain of the lumbar vertebrae whose normal columnar contour is altered to a wedge shape. The umbilicus is much lower than it is in the normal infant who stands erect. There is typical deformity of the hands and feet more apparent in the hands, which are short, broad and thick. The fingers are conical, and in the relaxed position of the hand the third and fourth finger tend to separate, producing a "trident hand;" the thumb constitutes the first branch; the second and third fingers, the second; and the fourth and fifth, the third branch of the trident. In most instances of this deforming disease, the humerus is short, with the result that the finger tips do not reach below the level of the femoral trochanter. This is not true in all cases, however, for in many achondroplastics the bones of the upper arm are not involved in the morbid process. The appearance of the head is one of the features of the affection, although an achondroplastic may have a perfect skull. The head is large and broad with a full forehead and well developed facial bones. In comparison with the vertex, the face is small, the nose is flat and depressed; and oftentimes the lips and tongue are thick, while in early infancy the tongue may be kept protruded. It is this latter peculiarity that sometimes makes it difficult to differentiate the face of an achondroplastic baby from that of a young cretin. The morbid process has no effect upon the eruption of the teeth which appear at the normal time, but the teeth them-

selves are apt to be irregular, and, as they develop, to be set widely spaced in the jaws. The general health of most such children is unimpaired and but few of them show any mental deterioration.

During the early months of life, it may become necessary to differentiate achondroplasia from cretinism or from osteomalacia, and very occasionally from Mongolism. In later infancy confusion may sometimes arise between achondroplasia and rickets. A careful inspection of the skeleton will reveal the typical short proximal limb segments and the characteristic bony changes of the head, neither of which appear in cretinism, and the cretin will usually show fat pads in the supraclavicular region with an undue degree of apathy, a subnormal temperature, and an obstinate constipation. In a child with osteomalacia, the distortion of the long bones occurs not at the epiphyses, as it does in achondroplastic patients, but along the shafts of the bones, and babies with the former disease have the bones of the cranial vertex imperfectly ossified in contrast to the normal ossification in the flat cranial bones of the latter group. The bony defects of rickets are many, and the general health of the patient is poor. Rachitic children are usually anemic and they suffer from malnutrition, while the general health of the achondroplasic is good, anemia rare, toxemia absent, and the bony deformities confined to the typical shortening of the long bones involved.

So far no *treatment* which in any way influences the deformities of achondroplasia has been devised. Thyroid extract has been given with the hope that it would influence growth and development in the cartilages that seem deficient, and a polyglandular therapy has been advocated. There seems to be no warrant for the hope that treatment in postnatal life can be effective, for the conditions encountered then are probably merely the result of an acute process developing in utero before the sixth month after conception.

Tuberculosis of the bones is very rare in early infancy and not at all frequent before the end of the second year. Tuberculous inflammation of the vertebræ and of the osseous structures near the hip joint provides most of the cases in late infancy, although the bones of the hand or any other bone may be subject to attack. Tuberculosis at this age, however, is prone to become generalized and exhibit a fatal form rather than to localize in the osseous system or anywhere else. The symptomatology and treatment are

discussed in the paragraphs on tuberculosis, chapter on Infectious Diseases.

Congenital syphilis attacks the bones of infants. Some writers believe the bony lesions of this malady to be as frequent as the cutaneous, and this may be so if all the minor manifestations of **osseous syphilis** are taken into consideration. The epiphyses of the long bones are most affected, and the cartilage and the neighboring periosteum are involved. For some reason, the epiphysis at the upper end of the humerus is that most often attacked. Syphilitic humeral epiphysitis is bilateral, although one arm is much more severely involved than the other. It is usual for loosening and separation of the epiphysis to follow the inflammatory process, although periostitis and softening may occur without any damage to the cartilage.

It is this manifestation of congenital syphilis which gives rise to the so-called *pseudoparalysis.* Palpation reveals swelling and tenderness at the site of the epiphysis. The arm is held flaccid and motionless, any attempt at movement is resented, and it is obvious that the pain and tenderness are extreme. A radiogram gives a characteristic picture which shows periosteal infiltration and, if separation of the epiphysis occurs, a widening between the cartilage and the shaft of the bone. The constitutional evidences of syphilis and the Wassermann complement-fixation reaction confirm the diagnosis in the presence of a pseudoparalysis with tenderness about an epiphysis. A similar pseudoparalysis with epiphyseal separation may occur in rickets, but the absence of the constitutional symptoms of lues and the presence of the clinical signs of rickets ought to render confusion improbable. The local signs that appear in the course of an *epiphysitis with scurvy* are more like those described, but here again the absence of the general evidences of congenital syphilis together with the symptoms indicative of scorbutis should make an accurate diagnosis readily possible, and the pathognomonic appearances of the osseous changes of scurvy on the x-ray plate will dissipate any doubt that remains.

The *treatment* apart from fixation to alleviate the pain of unnecessary movement, is that appropriate for the constitutional symptoms of syphilis. The use of mercury in doses proportioned to the age and weight of the child, whether it is given as inunction, injection or ingestion, will be followed by prompt relief. The use of arsphenamin in selected cases is a procedure of choice.

Apart from separation of the epiphyses, **congenital lues** is responsible for an occasional instance of **multiple fracture** during the period of infancy. The condition may simulate osteogenesis imperfecta and, when it does, it is usually associated with blueness of the sclerotic membrane. Often the same patient exhibits syphilitic fragility of the bones and a craniotabes due to atrophy of the bones of the cranial vault.

Dactylitis of syphilitic etiology is one of the rarest bone affections, but when it does appear, it is seen within the first two years of life, sometimes within the first two months—a time when tuberculosis of the hand bones is not seen. It is a malady diagnosed more often than its frequency warrants. The appearance of the periosteal shadows cast by the hand bones in syphilis and tuberculosis are sufficiently alike to render accurate differentiation difficult even for the most experienced radiographers. It is stated that the syphilitic affection is less apt to attack the metacarpal and metatarsal bones, but no reason has ever been assigned for their supposed immunity. The fact that syphilis of the bones may occur without any other sign of syphilis tends to accentuate the confusion. It may be said, however, that dactylitis occurring within the first year will probably prove to be syphilitic, and after that the differential diagnosis must rest upon the clinician's ability to determine concomitant symptoms either of syphilis or of tuberculosis.

Malignant growths, particularly **sarcoma,** have been encountered during infancy, sometimes at a very early time in that period. When such a growth does appear, it usually comes in one of the bones of the pelvic girdle, most often at the upper end of the femur. The swelling, tenseness and tenderness may simulate the local appearances of suppurative epiphysitis, but fever is seldom a feature until the terminal stage of the disease develops. The treatment is surgical but without any adequate results.

Multiple exostoses may be present when an infant is born, or they may appear at any time during infancy or childhood. They are apparently due to the lodgment of aberrant rests of cartilage. The affection is evidently a familial disease transmissible through either affected or unaffected members of the family, male or female. The tumors arise in bones formed in cartilage near the epiphyseal plate. Most of the outgrowths have occurred at the lower end of the femur, at the lower end of the radius, or at the upper end of the humerus. They may be present at birth and

escape detection by ordinary means of examination and be discovered by accident through the medium of a radiogram taken without any thought of their presence. On the other hand, the tumors may attain a considerable size in the period of antenatal life. They continue to grow throughout infancy until the final union of the epiphyses. For the reason that they increase in size at the expense of the other tissues, pressure may bring about muscular atrophies with weakness, or varying degrees of pain may follow the pressure effect of exostoses on neighboring nerves.

Arthritis in an infant is usually of the purulent variety, coming on as part of a suppurative epiphysitis. Apart from injury, scurvy and hemophilia, nonsuppurative arthritis is practically unknown, although a few young infants develop syphilitic inflammation of the joints complicating luetic involvement of the epiphyses. In these rare cases, almost always there is some development of pus which may have been secondarily infected but which in most of the reported cases has proved sterile. The toxic and chronic arthritides, so commonly seen in adults and sometimes met with in much older children, occur during the first two years of life only in the rarest instances. Such instances are limited to the very few cases in which **rheumatoid arthritis (Still's disease)** makes its appearance toward the end of the second year of life. In such a case low fever, with painless, symmetrical swelling of the joints, especially the finger joints, appears, together with palpable increase in the volume of the spleen and liver, muscular wasting, anemia, and leucocytosis, while evidences of syphilis and tuberculosis are entirely absent.

Rheumatic nodules, while common in older children, are rare under two years.

Occasionally a transient swelling in a single joint appears in an infant, together with other manifestations of recurrent *giant urticaria* (*angioneurotic edema*). It occurs, like the mild arthritis which may follow injections of heterologous sera, as a medical curiosity. Undoubtedly in both instances, the arthritis is but one evidence of an anaphylaxis.

Suppurative arthritis may result from the invasion of a joint by any infective organism. In infancy, the pneumococcus is by far the most common offender. The frequency with which the pneumococcus invades the blood stream of babies without producing a pneumonia is well known, and in more than half the cases of pneumococcus epiphysitis with arthritis there is no involvement of the lung. Otitis media, pharyngitis, or tonsillitis may

precede the appearance of suppuration in the joint or this may develop without any demonstrable antecedent or concomitant infection. In young infants, the hip joint is most commonly attacked; then in order, the knee, shoulder and ankle. The distribution is usually monarticular although it may be multiple. The symptoms are pain, tenderness, immobility, and an assumed posture calculated to relieve tension in the joint. Except in the early stages of arthritis, swelling is rarely limited to the region of the joint but invades large neighboring areas. Until late in the course of the disease, the overlying skin is not involved and shows no redness. Such a local pallor, taken together with the other symptoms, is suggestive of pneumococcus infection in the affected joint. The constitutional manifestations of the infection are few, much less than the observer would expect from the amount of local involvement and much in contrast with the appearances of extreme illness that are seen when the arthritis is of streptococcus or staphylococcus etiology.

The *streptococcus* is found the cause of infantile arthritis almost as frequently as the pneumococcus, but the staphylococcus, which is also causative, is a much less frequent offender. These organisms appear in the vascular areas about the epiphyseal cartilages and localize themselves to form small abscesses which penetrate the tissues and enter the joint cavities. Very often such localizations occur in the course of a generalized infection or a pyemia following invasion from any infective focus. As part of a pyemia, the outlook for recovery from a suppurative arthritis during infancy is bad, especially when the streptococcus is the invader. About half of the cases brought about by the pneumococcus recover under proper treatment which is essentially surgical. Temporizing measures, such as aspiration and fixation, are to be deprecated. Incision sufficient to insure adequate drainage is a life-saving measure to be adopted at the earliest moment after diagnosis has become possible.

As a medical rarity, an arthritis may complicate gonococcic *ophthalmia neonatorum* or result from a *gonorrheal vulvovaginitis* acquired at birth or developed during the early years of life. In those cases that have been reported, the arthritis has appeared from 10 to 20 days after the onset of the conjunctivitis. The inflammation may be confined to one joint or it may appear in several. Manifestations are usually limited to inflammation, swelling and tenderness without suppuration, although pus does appear

in a few instances, and when it is a feature, the process moves rapidly to a culmination. The joint distends and flexes, becomes acutely tender, and the patient evidences extreme pain on movement; the pressure of the swelling reddens and tenses the skin over the affected articulation, and the child shows symptoms of extreme constitutional involvement with a very high temperature.

Treatment of the milder cases is naturally to insure the proper management of the primary focus of infection, the eye, or vagina, by promptly applied local measures, and to relieve pain by the application of the ice bag to the affected joints, or by hot applications if these are better borne. When suppuration is obvious, early surgical treatment is indicated, for incision and evacuation may prevent destruction of the joint and a subsequent crippling. In a few cases which had become subacute or had shown a tendency to chronicity, an autogenous gonococcus vaccine has proved of value. The treatment of the results of a gonococcus arthritis is of necessity orthopedic. Physical therapy, hot applications, massage and passive movements are all needed in an attempt to restore function to the damaged joint.

Dislocation of the hip appears as the most frequent of all dislocations of congenital origin. It is very frequently hereditary and is found in several members of the same family. Girls are much more often affected than boys; in the proportion of 7 to 1. There is a slight preponderance of its occurrence as a unilateral condition. The etiology is still obscure. The condition has been found in the fetus. Certain of the theories which premise a defective development of the acetabulum or of the head of the femur are discredited. It has been considered, and this theory seems the most logical, as "an insufficient adaptation to an anthropologic fact of comparatively recent date," (erect position and gait as the race has advanced from the progression of quadrupeds).

The symptomatology in infants is somewhat obscure and is frequently not determined until the child begins to walk. At this time there is noticed a distinct disability and, if the dislocation is bilateral, a perfectly typical waddling gait with an accentuation of the lumbar lordosis. There is in the unilateral cases shortening and a lowered gluteal fold, and the trochanter is found to be below Nélaton's line. *Trendelenburg's* sign is positive; it is elicited by having the child stand on one leg, in which case

when the opposite leg is lifted, the pelvis is tilted downward if standing on the dislocated leg, and upward if standing on the sound leg. There is frequently a history of late walking, and there may be pain from the stretched sciatic nerve. A radiogram should always be taken, but must be interpreted by an experienced observer because of the cartilaginous condition of the bony parts at this age which renders difficult the determination of the true relationships. Differential diagnosis is as a rule not at all difficult. The **coxa vara** of rickets need hardly be confused with dislocation of the hip if careful observations are made as to the position of the trochanter and the head of the femur. **Coxitis** is rare in early infancy and there will be signs of inflammation. Similarly, paralytic dislocations, infrequently met with in early infancy, are rarely such that they are apt to be confused with hip dislocation. The various muscular atrophies, dystrophies, and spastic paraplegias have such distinctively neuromuscular symptoms and signs that they are readily differentiated. The prognosis is good—there is no spontaneous cure.

Treatment offers two methods, the open and the bloodless. The latter has been typified for a number of years by the method of Lorenz. This consists in a manipulation of the hip with reposition of the head of the femur into the acetabulum and retention by the aid of plaster of Paris. Success depends upon the continued apposition of the head of the femur in its normal relation for a period of at least six months; even then a recurrence may take place. It is wise to observe the result of the manipulation by radiograms. There is a recent observation of Frauenthal of New York which is of great value in the treatment of cases discovered early. It is not applicable to children who have walked for any length of time, and he has never succeeded with it in children over two and one-half years of age. Success rests upon the fact that the child has not borne weight upon the affected limb. This brings into question the disputed point which leads back to the etiology of the disease; namely, as to whether there is a congenital or acquired malformation of the acetabulum and head of the femur, or whether these structures were originally entirely normal. In such case, malformation is due to the fact that weight having been borne with a resulting stretching of the muscles and ligamentum teres, failure of function of the artery to the head occurs and finally atrophic changes.

Congenital club feet may be of the types, calcaneo valgus or equinovarus. In the former the dorsum of the foot is folded against the outer side of the leg while the heel points downward and the sole of the foot outward.

The *treatment* consists of simple stretchings and manipulations; these can be done by the mother or nurse under the direction of the physician.

The equinovarus type of club foot however, demands early and long continued treatment. This should begin within the first week of life when the baby's foot should be manipulated, turning the forefoot and toes outward into a position of pronation, the foot forced upward, thus stretching the calf muscle. This manipulation can be done without anesthesia and should be continued for fifteen minutes. After this the foot and leg to a point above the knee should be encased in a plaster of Paris cast so applied that as the bandage unrolls it produces tension to correct the deformity. The cast should be removed and replaced every two weeks; each time the tension and the amount of correction are to be increased in a direction of pronation and external rotation to correct the tortion of the tibia. This procedure must be continued until the child walks at about a year. Then if proper correction has been obta'ned, an internal bar brace with shoe attached, carrying a strap over the ankle should be applied. This shoe should be worn day and night. Daily massage and manipulation are to be continued. The heel of the shoe should be wedged on the outer side. If the brace is not worn for at least a year, relapse is certain. Even so recurrence is possible which may make it necessary to cut the plantar fascia and the posterior part of the capsule of the ankle joint, but not the heel cord. Markel calls attention to the error so frequently made, of dividing the tendo Achilles in which event the deformity remains uncorrected although the heel may be brought down.

The treatment of club foot and other like procedures should be referred to the orthopedist when possible.

CHAPTER XX

INTERNAL SECRETIONS

Present knowledge regarding the ductless glands, permits no doubt that these structures exert essential influence on bodily development and nutrition. As to the manner and method of the mechanism by which these influences develop, little is known, save in the case of the thyroid; but as Swale Vincent once cogently remarked, "more, much more is being taught about this subject than is known."

Aside from knowledge that the separate glands influence processes of development and growth, there is much certain evidence to indicate interdependence of these structures one with the other and with other glands and organisms of the body. Unfortunately the brilliant results that have followed thyroid feeding have led to the therapeutic abuses of many other gland substances which are administered without warrant to meet a variety of fancied pathological states.

During infancy, aside from the symptoms caused by hypothyroidism and by adrenal tumors, the symptoms produced by alteration in the internal secretions are vague and difficult of recognition. This is only natural, because the influence of these internal secretions weighs most upon manifestations of normality in growth, a process in itself deliberate in course and slow in expression and one which continues long after the period of infancy. The result is then that the more notable symptoms of disturbance in the function of these ductless glands remain in abeyance until the period of childhood, early or late. During the first 2 years, it seems logical to consider that these glands are as active as they are at any other period of life; perhaps more so. As to the thymus gland, its relatively large volume at birth and its progressive diminution during childhood, give us the right to believe that its activities are much concerned with the changes of this period, although there is little deducible from the results attained by experimentalists to sustain such contention. Similar deductions seem warrantable with regard to the adrenal gland, especially the cortical part of that organ, for it is a well demon-

strated clinical fact that changes in this tissue produce precocious obesity, great muscular development, with premature sexual characteristics either primary or secondary or both. The same sort of evidence applies to the pituitary gland whose influence on osseous growth and on the production of gigantism, seem to be demonstrated facts. The fact that certain tetanies are referable to pathology of the parathyroid glands seems undoubted. So far as changes in the other internal glands are concerned, the evidences of their disturbances appear so infrequently at any age that it is unnecessary to describe them in this connection. One of the striking features of alteration of body habit produced by changes in glandular secretion in infantilism, but as an infantilism is best defined as a retardation of the rate of development with retention of childish characteristics beyond the period of infancy, its discussion has no proper place here.

The **thyroid gland** may be quite markedly enlarged, producing fullness and even a small goiter without any evidence that the condition is giving rise to disturbances of the function of the gland. As a matter of fact, during this age, **hyperthyroidism** is practically unknown although it has been reported in children of between 3 and 5 years. As a medical curiosity, there is an occasional report of a child with a congenital exophthalmic goiter born to a mother suffering with the same disease; this condition has proved promptly fatal in all the reported cases.

On the other hand, **hypothyroidism** is a state that is common. To those who are interested in the subject and alert for its manifestations, the more marked degree that we call **cretinism** may be evident at birth or shortly afterwards. The infant cretin is apathetic; it usually is well supplied with coarse hair but as the condition progresses there is a tendency for the hair to fall out. The patient is adipose with the fat distributed in a manner typical of myxedemics of all ages—that is in aggregations about the supraclavicular triangles, under the arms, back of the neck, on the dorsums of the hands and feet. During this infantile period, the arms and legs seem short for the length of the body and the head and belly overlarge for the individual. The facial expression is characteristic even very early. The orbits are set far apart, the nose is flat and broad and thick, the palpebral fissures are narrow and they are often set at a slight angle; this is the only excuse for the common error that confounds cretins

with Mongolian idiots. The lips are loose and unnaturally thick. The same characteristics usually affect the tongue, which very frequently is so large that it cannot be contained in the mouth. The mucous membranes of the tongue, like the skin of the body, are coarse and there is a tendency to a thickening of the mucous glands and of the epithelium with resultant lingual fissuring. One of the most essential features of the clinical picture is the short thick neck; and even more revealing is the hand which is stumpy, with broad, short fingers, almost uniform in length, that tend to fall apart somewhat like the achondroplastic hand into a trident arrangement of the fingers. The circulation is poor, the extremities are apt to be mildly cyanotic and cold. Because of the influence the thyroid activity has in maintaining the level of metabolism, the temperature of these children is subnormal and they are readily affected by the cold. For the same reason, the pulse is slow and obstinate constipation is an almost constant feature of the disease. Although in extreme cases, the condition may be apparent at birth, in some instances the early signs may not be discernible until the fifth or sixth month of life; however, if the child is under careful observation, it is probable that suspicion will be aroused long before that time.

It is important that every apathetic baby be carefully studied, because the earlier the treatment can be instituted in these conditions, the more certainly favorable will be the result. As these babies grow up into the second half of the first year and into the second year, they are noted to be mentally dull; sometimes they show so little reaction to environment they may be classed as aments. As they begin to develop motility, the movements are slow and very deliberate. The acquisition of speech is delayed in time; and the vocabulary is greatly limited even in mild cases, and in the more extreme instances, it frequently consists of no more than a few syllables such as a very much younger infant may acquire.

The diagnosis of an even moderately developed case of cretinism is one that should offer no difficulty. The much more usual condition, Mongolian idiocy, is sometimes diagnosed as cretinism. The appearances of the former condition are pathognomonic; therefore, this mistake seems unwarrantable. There are, it is true, certain slight resemblances in the expressionless eye with its narrow palpebral slit and the enlarged and often protruded tongue which is common to both conditions. The typical smooth inner canthus

of the Mongolian eye is lacking, the shape of the tongue is quite different, thick and flat in cretinism but pointed and less immobile in the Mongolian. Apart from the eyes, the shape of the head of the two conditions provides a most essential difference. The Mongol's head is short, thick and broad with a flat occipital region. The Mongolian hand with the in-curved little finger, with its fine tapering digits, especially with its extreme hypotonus which permits the dorsum of the hand to be extended against the forearm, should be differential. The ears of the Mongol also are unmistakable in their typical deformities.

During the first half-year of life, it may be difficult to determine whether a given child is an achondroplasiac or a cretin. The short limbs, large head and flat nose are common to both, and it will not be until the observer has had an opportunity to determine the degree of intellectual alertness, and the character of growth in the femurs, that a diagnosis can be made.

Hypothyroidism in most instances is of congenital origin but in one of the writer's records is the history of a case in which the patient developed an abscess in the thyroid gland during the second year of life. There was evidently a complete destruction of gland tissue, for when the child was seen for the first time during its sixth year of life, its condition was indistinguishable from that of an extreme case of congenital cretinism. However, the patient, now eighteen years old, acquired perfect intellectual normality under thyroid therapy, and made usual progress through the grammar and high schools of San Francisco—an achievement which one doubts possible to any congenital cretin, no matter how well treated.

Acquired myxedema of varying degree is undoubtedly commoner during childhood than was formerly believed. However, the evidence of this condition appears only later in childhood and does not concern the purpose of this book.

As has already been emphasized, the degree of success in *treatment* will depend on the early recognition and prompt beginning of thyroid feeding. Every year of neglect involves a loss of time which results in a retardation in development which cannot be repaired. It is essentially during the years in which maximum growth and development occur in the normal child, that the cretin should be kept under the influence of thyroid extract. It must be recognized however, that very few, if any, of the children who are born with inadequately functioning thyroids will attain an absolutely normal intellect and physique, although the

physical status of the little one is much more amenable to treatment than is its intellect.

The dose of thyroid gland essential to the needs of the individual must be determined in each case. Insufficient dosage is undoubtedly a factor in the failure of the treatment. There are many cretins who will respond but slightly in intellectual and physical development, to dosages that disturb the metabolism and the nervous system to such a degree that they have to be lessened or temporarily omitted. Thyroid extract is by no means a harmless drug and with this knowledge in mind, it must be used with great caution. A dose that may be entirely suitable to one child may be too large for another and cause fever, irritability of the nervous system, restlessness and sleeplessness and in some infants, attacks of uncontrollable screaming. In little babies, it is best to begin with doses of $\frac{1}{10}$ to $\frac{1}{4}$ grain twice a day and to increase the quantity gradually, meanwhile watching for evidences of constitutional disturbance and of an increase in the basal metabolism. Most infants who are cretins will take 3 to 5 grains twice daily when the weight is 15 to 20 pounds, without any evidence of disturbance, provided this dosage is attained by gradual increases through a period of 6 weeks to 2 months. Some European clinicians advise the continuous daily use of the gland extract. In this country, it is more usual to give the drug for periods of 20 to 25 days with an omission for 5 days in order to watch the course of the disease and the effect of possible overmedication.

When thyroid medication is given to infant cretins early, and continued in adequate dosage, there is nothing more gratifying than the prompt change of some of these babies from revolting little automatons to smiling and often attractive and lively children.

The most acceptable investigations seem to indicate that the **parathyroid glands** are part of the thyroid apparatus and without specific function. It is true that certain animal experiments suggest a control of the parathyroid secretion over calcium metabolism, and for this reason tetany of infancy is thought by some observers to be an expression of parathyroid insufficiency. This view rests on the assumption that there is a calcium deficiency in cases of parathyroid disease.

Howland and Marriott have shown that in tetany there is a marked decrease of the calcium content of the blood serum. On

the average, their cases showed 5.6 mg. of calcium to 100 c.c. of blood serum as compared to finding of 11 mg. in normal patients. These observers are convinced that calcium deficiency alone is not the cause of tetany, as this syndrome does not occur in many cases of nephritis in which the calcium of the blood serum may fall as low as 1.5 mg. per 100 c.c.; their observations left these investigators unsatisfied as to the relationship of the parathyroid glands to calcium default and to the production of the symptoms present in tetany.

There is much contradiction in the deductions of various experimenters who have attempted to determine **thymus gland** function. What evidence there is, seems to point toward some partnership of influence with the genital, the thyroid and the lymphatic glands. Claims that were made by earlier experimenters that the internal secretion of the gland had marked influence on skeletal growth; but the more recent work of Holmes, McClure and others indicate that more facts must be developed before our knowledge of this subject can be accepted as satisfactory. The subcutaneous injection of thymus extract seems to act without effect other than that produced by the injection of any protein solution of like strength.

The gland in a normal newborn may vary considerably in size, and in its extent and its relation to surrounding structures. The average weight of the thymus gland of the newborn is about 10 grams. This weight increases up to the end of the second year. After this time, the gland atrophies. Increase in the size of the thymus may be due to simple hypertrophy, and its diagnosis, except by the aid of the radiogram, may be difficult. The evidence of thymic enlargement is the presence on the radiographic plate of an increased triangular shadow lying above the heart's opacity with the base of the triangle about coincident with the upper border of the sternum. The proper interpretation of the x-ray plate requires experience.

Apart from the x-ray, the so-called card sign method is the most useful diagnostic measure in delimiting the thymic area. In using this means, the stethoscope is placed high up on the manubrium and beginning near the edge of the stethoscopic bell, the skin is stroked with the corner of a calling card; the sweeps, parallel with the sternum, should approach the axilla. As soon as the air-containing edge of the lung is reached, a distinct change in the auscultatory tone is noted. This change in

sound rather accurately marks the limits of the thymus gland when confirmed by radiographic pictures. This evidence may be corroborated by simple percussion but such findings are not of great value. Even in very young babies, occasional masses of enlarged bronchial glands may impinge upon the mediastinum and confuse diagnosis.

In certain cases in which the thymus gland is enlarged, a striking symptom-complex, *thymic asthma* occurs; that this phenomenon is not due to the enlargement of the thymus alone becomes evident when one remembers the great number of children who come to autopsy in whom the thymus is much enlarged, but who during life showed no evidence of change in the organ. The authors on one occasion autopsied a Mongolian idiot, dead of pneumonia, who during life had not the slightest sign of disturbed thymus, yet a gland weighing 65 grams was removed in the course of the pathologic examination. It is also true that in many cases of thymic asthma and of the so-called "thymic death," the thymus has not reached such proportions as in the instance just cited. However, the majority of observers still believe that pressure is the cause of the paroxysms of thymic asthma, because the results of treatment by thymectomy or therapeutic roentgenology, especially the latter, is so completely successful in most instances.

In the clinical picture attributed to enlargement of the thymus, stridor is a dominant and unforgetable feature. Both phases of respiration are affected but inspiration much more pronouncedly than expiration. The stridor may become apparent a few hours after birth or it may not be heard for some weeks or even months. The degree of the stridor is variable; sometimes it is paroxysmal. It is rare however, that the larynx is obstructed or that the sound of the cry is much altered. This is especially true in those cases in which the stridor is continuous. On the other hand, when the stridor is paroxysmal, dyspnea may become intense and respiratory obstruction so great that extreme cyanosis is produced and if the paroxysm continues, death may occur from asphyxia. It is this asphyxial exitus that has constituted "thymic death" in the greatest number of reported cases.

These attacks may be produced and aggravated by overextension of the child's head and they are sometimes brought on by an angry child who throws his head back as an expression of his rage. However, that in certain cases, there is a toxic factor can-

not be doubted, for paroxysmal contractions of the larynx may be accompanied by dysphagia and by a widespread spasm of the bronchial musculature giving rise to auscultatory phenomena, indistinguishable from the sounds to be heard during an attack of typical asthma.

Thymic asthma is so characteristic that once seen, it is unlikely to be mistaken. *Congenital laryngeal stridor* is the condition most often confused. However, careful attention to the sound of the stridor will reveal that in the congenital type the noise is entirely confined to inspiration. There is never cyanosis and never any sign of respiratory distress and there is nothing paroxysmal about the condition. The only other source of stridor that might cause doubt would be the presence of a *papilloma of the larynx.* In this case, the voice changes are marked and it is almost unknown for the growth to attain sufficient size during the first 6 or 8 months of life to cause perplexing symptoms. A radiogram will distinguish an enlarged thymus and thus aid in differentiating it from laryngeal spasm or a papilloma.

Once the diagnosis is made, *treatment* is easy. Modern medicine has abandoned operative procedures or medicinal measures in favor of roentgenotherapy. This consists of subjecting the child to one-half an erythema dose of x-rays applied weekly for three or four weeks to the skin overlying the enlarged thymus. This form of treatment is thoroughly effective and improvement is usually prompt and the progress of the cure uninterrupted.

The relation of the thymus gland to the lymphatic system and to the so-called **status lymphaticus** is a matter about which little is actually known. Certain children who die suddenly and unexpectedly in the course of what is apparently a mild illness, during an anesthesial unconsciousness or without any apparent previous departure from health, are found after death to have been the possessors of hypertrophic thymus glands. They may also have a general enlargement of the lymphatic tissues, especially of the Peyer's patches and of the solitary follicles of the small intestine. All or only certain groups of the various lymph glands of the body may be enlarged. Sometimes the spleen exhibits a marked hypertrophy and at other times it is hardly increased in size. The lymph tissues of the faucial ring also share in the lymph hyperplasia. Fatty degeneration of the liver and hemorrhage into the serous membranes of the body

are almost uniformly discovered. The enlargement of the thymus in these cases has usually remained undiscovered until autopsy, and curiously enough, there is no record that any such child was subject to thymic asthma or stridor during its lifetime.

As this condition is rarely suspected during life, obviously there is no considered *treatment.* However, if in the course of a general examination a large spleen, general lymph enlargement, anemia, and excessive, flabby fat leads to the discovery of an enlarged thymus, it would seem reasonable to subject such children to roentgen therapy of this gland; and if the spleen also is overlarge it is advisable to treat this organ in the same manner.

The subject of affections of the **adrenal glands** is discussed in the chapter on Genitourinary Diseases. Congenital absence of the gland coincides with gross developmental defects, particularly anencephaly. The *malignant hypernephromas* are rare in infancy, though they do occur. *Addison's disease* has not been reported as occurring in a patient under 2 years of age.

During infancy, symptoms of **pituitary disease** will be in abeyance. Toward the end of the first year or during the second year, evidence of *osseous gigantism* may begin to appear in those rare cases in which pituitary secretion is overactive. *Acromegaly* is believed to be an evidence of hyperpituitarism but the changes in the bones and the enlargements of the lips and jaws do not become noticeable until later in childhood. The same may be said of *"Frolich's syndrome,"* the condition which follows hypopituitarism, in which there is marked hypoplasia of the genitals and the deposition of fat about the abdomen and breasts.

Disorders of the **pineal gland** which produce precocious mental and physical development particularly of the genitals, are not to be diagnosed during infancy.

CHAPTER XXI

INFECTIOUS DISEASES

Syphilis

Syphilis, as it appears in infancy, may have originated either in antenatal or postnatal life. By far the greater number of cases are of antenatal origin. The clinical manifestations of the disease fall into three groups, each dependent on the degree of virulence of the syphilitic poison. In the first group of cases, the virus is exceedingly potent and the resistance of the fetus minimal. Under these circumstances, abortion or stillbirth is the rule. In the second group, the poison is less severe and premature birth may occur; or the child may be born at term, alive, but with vital energy insufficient to permit its survival. Certain children of this group supply instances of acute fatal bullous eruption of the newborn, and also some of the cases of hemorrhagic disease of the newborn. In the third group, which provides most of the infant syphilitic patients, the influence of the poison is minimal and the resistance of the fetus high; therefore the child is born at term, apparently healthy, but later, sometimes between the third week and the third month, it develops symptoms that correspond to the exanthematous manifestations of acquired syphilis. The term florid syphilis is applied to the disease as it occurs in this group of patients.

The child who suffers from the **florid** type of syphilis is almost always the debilitated victim of malnutrition. Usually the earliest sign to attract attention to the condition is an acute semipurulent coryza which soon becomes sanguinous; and at the same time an erythema appears, together with excoriations about the nostrils and the corners of the mouth. At this stage of the disease, a smear of the nasal discharges, examined under the dark-field will usually show the etiological spirochete. Frequently the erythema changes to a deep-seated inflammation of the skin with excoriations of the epidermis and deep fissures which appear across the mucocutaneous margins of the lips to form linear ulcers (*rhagades*). As these heal, characteristic white contracted scars are left. This process must be sharply differentiated from the scars of cracked lips which are sometimes quite deep but

which seldom pass the mucocutaneous margins and rarely occur at the corners of the mouth, a site characteristic for rhagades. Simultaneously with the appearance of these circumoral ulcers, smaller characteristic *eruptions* may be found about the nates, especially about the anal orifice, where the proliferating, ulcerating masses of epithelium known as *condylomata,* pathognomonic of the disease, appear. About this time a generalized erythematous rash may be seen over the skin. Superficial ulcers of the mucous membrane in the mouth and on the fauces develop, although the latter are unusual manifestations. Most often the rash is limited to the buttocks, the face and the forehead. The rash about the buttocks has a reddish brown color and tends to spread beyond the napkin area. Except about the mouth and nates, the eruption is usually papular, although at times bullæ may appear; but on the whole, bullous eruptions are unusual, except in cases of **pemphigus neonatorum,** a condition which is almost always fatal.

As a complication of the florid type of infantile congenital syphilis, **epiphysitis** is frequent. It has been demonstrated in the prematurely born and in stillborn children, and like the rash, it is a result of intimate changes in the blood vessels following an invasion of the vascular coats by the Treponema pallidum. The epiphysis most commonly attacked is at the upper end of the humerus; and next in order, that at the lower end of the femur. The observer's attention is rarely called to the epiphysitis before the second or third month of life when pain, immobility and swelling at the site of an epiphysis manifest themselves. Immobility is such that it may quite perplexingly mimic a true paralysis. Palpation however, will reveal tenderness and a characteristic swelling. The protective reaction to pain is not always the cause of the immobility because in some instances there is no tenderness and apparently no pain. It is rare for more than one epiphysis to be attacked at the same time. Most often only one of the epiphyses is affected during the course of the disease. When the condition is very severe, separation of the epiphysis may result and then growth in the length of the bone is interfered with. Syphilitic epiphysitis is essentially a disease of the early months of life; and while it mimics scurvy (which rarely occurs during the first half-year) the age incidence of the two diseases is a great aid to differential diagnosis. Should an epiphysitis appear in the later months of infancy, the swelling is less and the

tenderness not so intense as in scurvy. Further aid is derived from the fact that the patient with luetic epiphysitis will usually show some other signs of syphilis.

Acute, septic epiphysitis must be differentiated. This is usually a matter of no difficulty. The appearance of the child, the tremendous pain and tenderness, the blood picture and the absence of a positive response to the Wassermann test in the septic disorder aid in the differentiation.

Even in early infancy, damage to the vascular system is by no means always the rule. Many children with congenital syphilis show no other signs than those following a profound disturbance of nutrition, anemia, wasting, anorexia and a toxic appearance. Such children should always be tested for the disease by the Wassermann procedure, in spite of the fact that complement fixation is an uncertain guide to diagnosis in the early months of life.

In the later periods of infancy toward the end of the first year, certain manifestations of syphilis appear which are comparable in their pathologic changes to those that occur in the tertiary stage of the acquired disease. These include **periostitis, meningitis, keratitis** and **ulcerative lesions of the skin.** They are essentially chronic and for the most part painless and resistant to treatment. Such expressions of the disease are infrequent before the second half of the second year, and even then they occur less often than in the subsequent periods of childhood. The bones of the hands and feet are commonly attacked with a type of dactylitis which is difficult to differentiate from tuberculous changes in the same structures. In some instances radiograms may be elucidative. The tibias are also favorite sites for this type of periostitis, and as a result of lesions here the saber-shin develops.

The same kind of an infiltrating process attacking the cornea gives rise to opacity with salmon colored injection, which is found only in **luetic keratitis.** The differentiation between the corneal infiltration of this disease and that of tuberculosis is not simple.

Fortunately during the first 2 years of life, syphilitic **basal meningitis** or involvement of nerve trunks is almost unknown; but Jeans has shown that in lues of the florid type, lumbar puncture reveals changes in the meninges akin to those that are going on in the skin, and he contends that the nervous system is involved in nearly all the cases of florid syphilis. This is in keeping with the observations of Comby who, many years ago, showed

that the skin changes of the exanthematous fevers are accompanied by inflammatory changes in the meninges.

There is some reason to believe that certain rare cases of *hydrocephalus* are due to a low grade meningitis of syphilitic origin which interferes with the absorption of the cerebrospinal fluid.

The profound influence of the syphilitic intoxication on growth brings on certain skeletal and dental changes, none of which is truly characteristic of the disease except the deformities of the permanent teeth **(Hutchinson's teeth)**. These are damaged while they are still in their germ stage. The lateral buds may fail to develop fully, producing the peg-shaped incisor; or the central bud may be dwarfed and a semilunar edge follow. Radiograms of the unerupted incisors may show Hutchinson's deformity present.

The *treatment* of congenital lues should begin in the prenatal period. The child should receive its medication by way of the mother's circulation. The parents' blood should be examined on slightest suspicion. The treatment of the mother should be begun promptly, even though the mother's blood should give a negative response, provided the father has a positive reaction or if there be any clinical grounds for suspicion. A negative Wassermann reaction both in adult and infant has not the significance of a positive reading. Some syphilographers attribute no value whatever to a negative reaction. Vigorous treatment of the mother has a double effect in that it tends to eradicate the disease in her and to influence the infant in utero through the maternal blood. Experience has shown that the incidence of abortion and stillbirth is much lessened in syphilitic pregnant women who have undergone treatment during their pregnancy, and that the probability of syphilitic offspring is greatly reduced. The treatment of such women differs in no way from the usual treatment of acquired syphilis in the adult. It has been shown that arsphenamine does not increase the tendency to abortion and hemorrhage, but on the contrary often prevents them. There is no valid reason why the pregnant woman should not be as intensively treated as though she were not pregnant. Neoarsphenamine, mercury and the iodids all have a place in such a program.

Children born with *syphilitic pemphigus* or with other well-marked, deep skin lesions, and who are in a state of extreme debility, respond very badly to treatment. Even if they recover from the lesions, they rarely become more than miserable children with poor expectation of life and health.

Syphilis is one of the diseases in which prompt treatment is well rewarded. In the commoner florid type of the disease, when snuffles appear about the sixth or eighth week of life, an immediate resort to neoarsphenamine and mercury is usually met by dramatic amelioration of the symptoms. This phenomenon, however, must not be mistaken for a cure.

Strictest attention to the details of feeding in young children is as important as the use of drugs. Veeder, in summing a report of 100 cases treated at Washington University, says that "the therapeutic results are disappointing as a whole" and that "the outcome of the case, as far as life and death are concerned, depends far more on the condition of the patient's nutrition than upon the character of the specific medication."

When mother's milk is of good quality and quantity, the infant should be nursed. It is obvious that a wet nurse who is not infected should not be used to suckle the baby in case of a failure of the maternal supply, although she may be advantageously a source of milk. The milk is best obtained from her breast by manual expression. Under this system healthy women who are not over nervous, can be taught to produce the maximum quantity. Human milk should be fed to the luetic infant in preference to any other food. In case it is impossible to get human milk, a well devised formula of cow's milk is next of choice.

Mercury is the time-honored drug for use in cases of congenital syphilis. It is unquestionably effective in eradicating the symptoms of the disease in most cases. It may be given by mouth, by injection, or in the form of inunctions. When given by mouth, in doses sufficiently large to be therapeutically effective, mercury has the disadvantages of often disturbing the digestion and producing diarrhea. If it is desired to administer the drug in this manner, there is no better preparation than mercury and chalk which is so commonly used in British practice. The usual dosage for a 15-pound infant is $\frac{1}{8}$ to $\frac{1}{4}$ of a grain four times a day. It is less liable to cause a diarrhea than any of the other preparations of mercury, but it may be the source of annoying abdominal pain.

Injections are painful, but when rapid results are urgently needed, they may be necessary. In such circumstances, injections may be given into the triceps or gluteus muscle. The former site is preferred because there is less chance of abscess formation there than about the buttocks. In case it is deemed advisable to use in-

jections, the soluble forms of the drug are of greater advantage and efficiency. The sozoiodolate of mercury given in 5 minim doses of a 1 per cent solution is thoroughly satisfactory. The bichloride of mercury is sometimes useful in doses of 1/200 to 1/300 of a grain for a 15-pound baby. It should be injected every other day for a period of 2 weeks. After a rest of 2 weeks a similar course should be repeated, unless the lesions have shown marked amelioration. After the lesions have shown a sufficient response to injections of mercury, one of the other methods of administration—inunction or ingestion—may be used.

The method of choice for the administration of mercury in infancy is inunction. The only objections are that the most effective unguent containing mercury, the blue ointment, is unpleasant in appearance and soils the underclothing. It has the further disadvantage of being recognizable to the laity, and its use may embarrass the parents. However, in the severe cases its superior efficacy makes its use imperative. There are preparations especially devised for inunction. These carry mercury in a bland cocoa-butter base and are free from some of the disadvantages of mercurial ointment. Prepared by reliable manufacturers, they can be used 20 grains at a time for the inunction of infant patients. Such a portion should be rubbed into the skin every second day. It is wise to vary the area of application from day to day using back, belly, chest, axilla, popliteal space and cubital fossa in sequence. The absorption is better if the inunction is made following a warm bath. The theory that mercury is not absorbed by the skin but gains entrance into the body through the inhalation of the metallic vapor has been disproved by Ramsey of St. Paul who finds it excreted in the urine when the mercury applied to the skin is covered by impermeable membranes.

In those cases in which the symptoms are less florid, white precipitate or yellow oxide of mercury carried in benzoinated lard may be used in 5 per cent or 10 per cent strength. The dosage is the same as for mercurial ointment.

Local lesions about the mouth and buttocks are better treated with powders or lotions than by salves. A very effective lotion is made by adding 10 to 15 grains of ammoniated mercury to 3 ounces of calamine lotion. When there are ulcerating lesions in the nates, a small compress saturated with this lotion should be laid between the folds in order to keep the opposing surfaces

apart. The same procedure should be followed when the lesions appear in the groins or axillæ. For use on other parts of the body the lotion should be shaken well, poured out into a small dish and then dabbed onto the lesions with cotton. Another lotion of value is made of equal parts of black wash, milk of magnesia and rose water. This should be applied on compresses or, if used about the face, under a mask.

For the dry crusted lesions with underlying ulceration, such as are seen about the eyebrows or on the forehead, unguents are better than lotions. Three per cent ammoniated mercury in lanolin soon softens the crusts, which can then be washed away with green soap; after this, a lotion should be applied.

There are commercial mercurial preparations on the market designed for inunction, some with a fatty, others with a soapy base. None of these has any advantage over a freshly prepared and well made mercurial ointment, and those with a soap base are sometimes irritating to the skin of an infant.

In the profession neoarsphenamine has provided a remedy that is more efficacious and less toxic than is arsphenamine. Dr. Hermann Schussler of Stanford University Medical School, has shown that infants tolerate neoarsphenamine particularly well. He advises average doses for babies under 10 pounds, .15 grams; 10 to 20 pounds, .3 grams; 20 to 35 pounds, .45 grams. The first dose to be given any child should not be more than half the dose computed as appropriate to its weight. Ormsby states that the required dose of this drug should be given to the infant 4 times with intervening intervals of 48 hours; in our practice this advice has proved sound. Few infants treated in this way show signs of cumulative effect. Following the fourth dose, triweekly inunctions of mercury shoud be given for a period of six weeks; then should follow a rest of four weeks. In the same way the course of 4 neoarsphenamine injections is to be repeated. This again is followed by six weeks of inunctions and four weeks of rest; then, for the third time, the 4 injections of neoarsphenamine are again given at 48 hour intervals; and these are for the third time, followed by a six weeks' course of triweekly inunctions. This procedure usually suffices to bring about a negative Wassermann reaction in a child whose treatment has been begun before it is 6 months old. For older infants a course of alternating injections and inunctions may have to be repeated from 6 to 10 times.

The neoarsphenamine for intravenous injection should be used in a 30 per cent solution, (.3 grams to 1 c.c. cold water). The use of such strength solution is followed less often by unpleasant reactions than when greater dilutions are employed. A 1½ c.c. glass, hypodermic syringe may be used for both the mixing and the injecting of the preparation. This size syringe may carry a 25 gauge needle which is ideal for puncture of the vein. During the first year the veins of the scalp are most readily accessible and may be used for the intravenous injection. In later infancy the external jugular vein is most easily entered.

Neoarsphenamine for rectal injection may be made into a 10 per cent solution, and after the primary enema, with the infant's hips raised, the solution at room temperature is slowly passed into the rectum through a small catheter. The rectal dose is one-half greater than the intravenous.

Acquired syphilis usually runs a more destructive and a more rapid course in young children than in adults. It is also more virulent than congenital syphilis in children of the same age. The *treatment* is in no way different from that outlined for congenital syphilis except that the mother must be protected from contact with the infant and therefore nursing at the breast is to be forbidden.

Tuberculosis

Tuberculosis is variable in its manifestations. Recovery from the disease, when it attacks the child during the first year, is almost unknown. In the few known cases where the patient has survived and a chronic tuberculosis has supervened, the mother of the infant has had an active tuberculous lesion during her pregnancy. Under these circumstances, the child acquired enough antenatal immunity to prevent the activities of the tubercle bacillus from becoming lethal. One such case is recorded at the University of California Hospital. In this instance, multiple bone lesions in numbers almost incredible, together with radiographic evidences of profound lung invasion, seemed incompatible with the course of the disease which appeared in the early months of life and ran on late into the second year.

On the other hand, when the mother is the subject of an active lesion during the later months of pregnancy, bacteria may pass the placental barriers and enter the infant circulation. Cases are recorded in which infants have died from generalized tubercu-

losis within a few days after birth. Sometimes the appearance of generalization is delayed for weeks, rarely for a few months.

More often, however, the mother does not infect her child antepartum. Children are born uninfected, but the tuberculous mother, because of close contact, becomes a dangerous source of infection. The view of Hamburger and von Behring, that all tuberculosis is implanted in infancy, while not definitely proved or completely accepted, is an hypothesis that must receive great consideration. This teaching, together with the fact that an actively tuberculous mother is a source of danger to her child, is a warrant for what may seem an inhuman practice—the removal of the baby from all contact with the mother. Hard as this may seem, there is no alternative for the conscientious practitioner. On the other hand, the obstetrician should see that the tuberculous woman is fully instructed of the danger to herself and to her child if she becomes pregnant.

The clinical manifestations of tuberculosis as it attacks infants in the first and second years are so variable that in the compass of a small volume they can be no more than indicated. Inability of the young child to resist the disease results in a rapid generalization of the process. During the first year of life, the preliminary stage of the infection is a bacteriemia which runs a course of some weeks, or perhaps of some months. It is revealed by a low degree of fever which rarely exceeds 103°; more often, the fever ranges between 100° and 102°, although an afebrile or a subnormal temperature curve is quite compatible with this stage of the disease. Pallor, anorexia, irritability, constipation, and malaise varying in intensity from time to time in the individual case, are characteristics of this early period. Accompanying these symptoms, malnutrition and loss of weight are slowly progressing evidences of the disease.

After such a preliminary period, somnolence of a greater or less degree sets in and sometimes, but not by any means always, headache develops and is revealed by the child's piercing, cephalic cry. In the later periods of infancy, headache is signalled by a characteristic boring of the head into the pillow or by the beating of the vertex or the forehead with the hands. In the presence of these signs, palpation of the head reveals bulging fontanels, increased widening of the sutures, and a characteristic, ripe watermelon sound on percussion over the parietal bones. At any time during the course of such a process, the child may begin

to vomit. The emesis becomes more vigorous and frequent as time goes on. Rarely, it is the most striking feature in the clinical picture. Such preliminary stages may last in some instances a few weeks, in others many months before the circulating bacteria attack the meninges. When the meninges are invaded, the child usually has a convulsion of greater or less severity.

Tuberculous meningitis in childhood is not a clinical entity but a terminal event in a generalized tuberculosis. Some days before the onset of the first convulsion and afterwards as well, signs of irritation of the brain and meninges may appear. The commonest of these is muscular twitching which affects the hands, face and limbs. This type of muscle twitching is characteristic of tuberculous meningitis. The observer may note a twitching of a finger, perhaps accompanied by a slight clonus of a group of muscles in the arm. This may cease, and a few moments later, movements about the mouth will be observable. After these have lasted for a little while, another group, perhaps in a lower extremity, may show passing clonic contractions. Most striking are the irritations and palsies of the eye muscles resulting in strabismus, ptosis and irregularities or fixations of the pupils.

Only one other disease mimics the nervous manifestations that appear during the early stages of a tuberculous meningitis, and that is an encephalitis which is differentiable by virtue of the history. The onset in encephalitis is sudden and fulminant; it lacks the typical twitchings of tuberculous meningitis and there is a difference in the course of the two infections.

As the tuberculous meningitis progresses, intracranial pressure signs usually become dominant; increasing lethargy, eye-ground changes and finally respiratory and circulatory disturbances become evident. Grouped breathing which develops late is a precursor of death.

At any time in the course of the disease, a radiogram of the chest may be of help in diagnosis. The mossy, mottled lung of miliary tuberculosis will be in evidence.

At the stage of onset of meningeal symptoms, lumbar puncture is confirmatory. Most often the fluid is found to be under considerable pressure; it is clear unless accidentally contaminated with blood. If a tube of the spinal fluid is set in a warm place, after a few hours a delicate clot or pellicle will be found hanging from the surface of the fluid. This clot has been called the "web" because of its similarity to the finely spun, reticulated web of

a spider. This web can be floated out, stained and the tubercle bacillus often demonstrated. The technic devised by Cheer is simple and effective. It consists of precipitating a cloud of coagulum by the addition of a small amount of 95 per cent alcohol. After centrifuging for 30 minutes a loopful of the precipitate is dried and stained with Ziehl-Neelson stain. The tubercle bacillus is usually demonstrable without prolonged search.

The first puncture will sometimes produce a fluid in which a web does not form. This is especially true when a puncture has been made immediately after the appearance of the meningeal irritative signs. Cytologic counts often reveal a high mononucleosis, although in rare instances this help may fail and both the spinal fluid and blood show, instead of lymphocytes, a large preponderance of polymorphonuclear leucocytes. This is especially true in cases of very acute onset. There is always an increase of globulin with a diminution of the carbohydrates in the fluid. Occasionally the spinal fluid from an encephalitic patient will give all the physical characteristics of the fluid of tuberculous origin. However, the fluid found in encephalitis almost always contains certain large mononuclear leucocytes which are pathognomonic.

The von Pirquet skin test as an aid to diagnosis, especially in the generalized forms of tuberculosis, is of confirmatory value. In a young child, the presence of a positive reaction is certainly suggestive, but the absence of such a reaction may be misleading as it fails to appear in many cases of generalized tuberculosis.

A very few cases of generalized tuberculosis with invasion of the cerebrospinal system terminate without having developed convulsions, twitchings or motor evidences of meningitis. The writers have studied such a case and demonstrated the tubercle bacillus in the spinal fluid. In this type of invasion, the spinal fluid is scanty and without increased pressure. These children die in coma with all the evidences of profound toxemia.

The *treatment* of generalized tuberculosis and of tuberculous meningitis, in the present stage of our knowledge, is limited. It must be assured that the woman with tuberculous lesions in her lungs, either active or quiescent, will not be allowed to bear children. When a child is born of a tuberculous mother, it should be removed at once from contact with her.

After the birth of any infant, scrupulous care must be taken to prevent the invasion of its environment by an actively tuberculous person. Maids, nurses, friends, and acquaintances have

all been carriers of the deadly organism to young children. While it is impossible to keep the child in monastic seclusion, the mother can be taught that there are many simple and sensible ways of protecting the child. The deadliness of the casual kiss must be recognized. The kissing of infants and young children should be abolished as a social habit. The rigid exclusion of every coughing individual from close contact with the child will prevent not only primary tuberculosis but coryza, laryngitis, or bronchitis—maladies that by diminishing the resistance of the infant may lay the foundation for tuberculosis.

The fact that most of the tuberculosis of little children arises from human contact, is no reason why we should omit the most careful supervision of our milk supply. Contamination of children by bovine tuberculosis is common. It may originate from ingested milk or by contact with some human who is infected with tuberculosis of the bovine type. The use of certified milk, or of properly pasteurized milk, or failing these, of boiled milk, will limit the chance of such infections. The physician can aid the veterinarian by preaching the gospel of tuberculosis testing for all dairy herds.

The management of a child suffering from a generalized tuberculosis is the same as for a child with malnutrition. Heliotherapy, cod-liver oil, careful attention to feeding and to general hygiene should constitute our endeavors in such circumstances. Once the involvement of the central nervous system has occurred, treatment of any sort is an unkindness except in those cases in which lethargy and somnolence are little in evidence and headache is distressing. In order to lower intracranial pressure and thus relieve the headache, the withdrawal of spinal fluid every 12 to 24 hours is a humanitarian measure. As much fluid should be drawn as will flow freely. If puncture does not relieve the headache, the administration of drugs is useless unless the patient be thoroughly narcotized. The continued use of spinal puncture is of no curative value nor is any other measure that human ingenuity has devised. Treatment intended to inhibit the convulsions and twitchings is most discouraging. Opiates and sedatives are almost useless. We must resign ourselves to keeping the patient as clean as possible and to feeding it as best we can.

Although tuberculosis as it occurs in the first year of life is for the most part a generalized disease, there are exceptional cases in which the process is obviously localized during the early part of

the attack, but later, goes on to generalization. In a large proportion of these exceptional cases, the site of localization is the mastoid region. Even as early as the sixth or seventh week, **tuberculosis of the mastoid** has been demonstrated. It may run 8 to 10 weeks before generalization becomes evident. The child will die sometime later with a terminal meningitis. The mild, insidious onset of such a mastoiditis, its slow progress, the high lymphocytic formula of the blood, the increasing anemia and malaise, together with the positive von Pirquet skin test, form a group of signs and symptoms hardly to be mistaken.

As in all other forms of the disease, the treatment is limited to the provision of as nearly perfect hygienic surroundings as possible. Surgical interference in these cases can do no good and may hasten the generalization of the disease.

Tuberculosis attacking the spine is rarely seen in the first or during the early part of the second year, but toward the end of the second year, it is encountered at times. Unfortunately, many cases of early **Pott's disease** are overlooked because of a failure to appreciate its earliest evidences.

It must not be forgotten that pain in the belly is an early, and often the only, sign of the invasion of the spine by the bacillus of tuberculosis. When the disease attacks the lower dorsal region, the pain is above the umbilicus. Too often a child is treated for colic or for indigestion when the real source of distress is in the spinal column. When the lumbar spines are involved, the pain is about or below the umbilicus. From carious upper dorsal spines, the pain is referred to the front of the chest and when the cervical vertebræ are involved, the pain may be felt in the neck, shoulders and arms and will be aggravated by movements of the head. Spasms of the muscles appear particularly early, if the disease has attacked the cervical vertebra. Wry-neck, developing during the second year of life, must be investigated as a symptom of a possible tuberculosis. One very suggestive symptom is exacerbation of pain on rough movement. For instance, if the child is being taken on a street car or in an automobile, inequalities of the track or road cause it to cry out with pain. This symptom, taken together with pain in the belly should lead to radiographic search for early spinal caries and if the search reveals evidence of bony involvement, it may be possible to forestall the progress of the caries and to prevent deformities by the use of heliotherapy and by the proper regulation of the child's diet and hygiene.

For a full consideration of the treatment of spinal caries and other destructive bone lesions and the deformities that develop from them, the reader is referred to standard orthopedic manuals.

Tuberculous epiphysitis most frequently attacks the upper femoral, sometimes the upper humeral epiphysis and rarely the epiphysis of other long bones. Infants, who are beginning to walk, may be attacked at the epiphyseal cartilages by tubercle bacilli; but it must not be forgotten that there are many other causes of epiphysitis, and it is unwise immediately to decide that any child with the symptoms of hip joint involvement has become a victim of tuberculosis. In their early experience as pedestrians, babies are subject to wrenches, twists and bruises through which their epiphyses may be injured, with a resulting lameness or disability which often will clear up, sometimes after a short rest in bed. At this age also, toxic and septic epiphysitis are not unknown. The symptom-complex of all these conditions is the same. The child limps a little, muscular spasm develops to protect the tender hip joint, and the leg is carried in outward rotation. Later, with an increase of fluid in the joint, inward rotation takes place. There is flexion of the knee and the limp increases. Attempts to place the heel of the foot in the opposite groin meet with resistance from the spasm of muscles and the maneuver is found impossible. Inspection of the back of the unclothed child shows that the folds of the nates on the affected and well sides are not in line. The blood count will sometimes be of value in aiding to differentiate conditions which are easily mistaken for very early tuberculosis of the hip. The final decision must be made after considering the results of rest in bed for a period of 4 or 5 days. After such a rest, the symptoms of an epiphysitis due to injury or toxemia should have disappeared. Roentgenograms are of positive value only when the process in the bone has begun to be destructive, although skillful radiographers can draw many deductions from the relations of the bones one to the other.

In tuberculous epiphysitis, rest in bed, with fixation, heliotherapy, attention to proper nutrition, and general hygienic measures are indicated. Whenever possible, the advice of a competent orthopedic surgeon should be taken.

The epiphysis of the upper end of the humerus is sometimes attacked by disease and the differential diagnosis must be made between tuberculosis, scurvy, syphilis, septic epiphysitis and in-

jury. The radiogram is of much more aid in elucidating conditions in this area during the early stages of the infection than it is when used to picture the hip joint under similar conditions. Tuberculous changes are commoner about the upper femoral epiphysis than nontuberculous, while in the region of the upper humeral epiphysis, the nontuberculous are more frequent. The rapidly on-coming symptoms of scurvy, as well as of syphilis and of sepsis, when they localize about the humeral epiphysis, are promptly followed by changes so extreme that they are easily registered by the x-ray plate. On the other hand, the tissue alterations that accompany the slower developing pathological processes of tuberculosis may be so slight that for a long time after function has been interfered with, radiographic evidences of the disease may fail to appear.

The treatment of tuberculosis of the upper humeral epiphysis calls for the same management useful when other epiphyses are attacked.

There are certain bones which seem to be especially vulnerable to tuberculosis during babyhood. From the last part of the first year through the second and third years, the small bones of the hands and feet, the carpals and metacarpals, the tarsals and metatarsals, become tuberculous with moderate frequency. This involvement is known as **dactylitis.**

Children suffering with this manifestation are, for the most part, fairly well nourished, and under these circumstances the tuberculosis organism is rarely virulent. The lesions are slowly destructive but they are self-limited and generalization of the infection is extremely rare. Although the periosteum is involved and raised and the overlying tissues greatly swollen and infiltrated, pain is not a feature. These peculiarities frequently lead parents to neglect the earliest evidences of the trouble, and the patient often comes to the physician only after widespread destruction of the bone has taken place. Often, considerable areas of the soft tissues have become involved with necrosis and liquefaction. Such a pathologic process results in a characteristic appearance. The spindle-shaped swellings of the fingers and toes, over which the skin is tense, shiny and red, reveal the invasion of the carpal, metacarpal and phalangeal bones. Irregular shaped swellings of similar appearance arise over the dorsum or sides of the hands or feet. Radiographic pictures at this stage are unmistakable, although in recent years some confusion has arisen

over the differentiation of this type of tuberculosis from syphilis of the bones. The fact that in tuberculous dactylitis there is much periosteal proliferation and extensive underlying destruction of bony shafts has been responsible for this confusion.

The *treatment* of the condition in the fully developed stage belongs to the orthopedic surgeon. However, it may be repeated that there is no method of treatment as efficient as heliotherapy. (See Methods.) Scrupulous care must be taken to provide the child with adequate nourishment which will be aided if cod-liver oil and phosphorus are used as adjuvants. It should be the concern of the general practitioner to recognize the early stages of these bony lesions and to initiate prompt treatment in order to prevent the development of extensive destruction. The first signs of this disease consist of slight tenderness of the fingers, a moderate stiffness of the joints together with a little thickening about the phalanges, and an unwillingness of the child to use the hands freely. These signs are often so trivial that they are easily missed, but it is wise to make a careful observation of the hands of every child who is under treatment. If a diagnosis can be made early and if it is possible to apply heliotherapy to the infant and properly to control its nourishment, a dactylitis may sometimes be aborted.

Apart from tuberculous epiphysitis, **synovitis** is rare in infancy. It usually occurs after the third year and is a slow, chronic and painless process. A radiographic picture of the chest will almost invariably show that the patient has a coincident lung involvement.

Ulcerative lesions of the skin of tuberculous origin occur in childhood. They are unusual and they seem of thrombotic origin. Destruction is limited to the skin, and it is probable that the pathologic process is closely related to that in lupus. Originally, the condition was described by Bazin as **erythema indurativum.** In the first stage of the inflammation, a red papule appears. This increases in size rapidly, and in 24 to 48 hours the destruction has gone on to the production of a large ulcerated surface. The only effective treatment is wide excision and subsequent skin grafting.

Lupus, which is common in adults, rarely occurs in infants, and its treatment need not be considered here.

Tuberculides appear on the skin as small, pale, raised papules, about the size of pin heads. Many of the papules are surmounted by a tiny necrotic area of brownish tint. Tuberculides rarely

appear except in the later stages of a generalized tuberculosis, and they are of little significance except as an indication of this condition. This eruption is sometimes accompanied by itching which may make the child's last days very unhappy. This itching can be alleviated by the application of lotions containing 1 per cent to 2 per cent phenol or 5 per cent coal-tar solution.

A general **erythema with hemorrhage** into the skin appears in some infants as a terminal event in the course of a generalized tuberculosis. It is a precursor of immediately approaching dissolution, and as such its treatment is immaterial.

It is an unusual experience to meet the adult type of **tuberculosis of the lung** in infants. When tuberculous invasion does take place in this way, the distribution of the lesions varies strikingly from that which occurs in the adult. In later childhood and in maturity, the lung apices are most often first involved. In infancy the middle right or one of the lower lobes is the site of predilection, and the spread of the pathologic process throughout the lung is quite rapid. The disease almost always ends fatally, usually with generalization.

The work of Gohn seems to have successfully contradicted the assumptions of the Cornet school that tuberculosis of the lung in infancy and in early childhood is secondary to a primary tuberculous adenitis and that infection for the most part is by ingestion. Making many serial sections of the lungs and studying them carefully, Gohn produced one of the most illuminating studies in the history of pediatrics. He was able to demonstrate with great precision the primary focus in the lung and to recognize what pulmonary areas drain into given groups of mediastinal glands.

The physical signs of **tuberculous lung involvement in older infants** are modified by the delicacy of the tissues; otherwise, they are much the same as those discernible in adult tuberculosis. There is a more obvious limitation of movement on the side of the affected lung; dullness is not so profound; and the auscultatory signs are more complicated. This complication is due to the presence of secretions in many of the larger bronchial tubes. In the nature of things, this bronchial phenomenon arises because the infant has little ability to empty the pulmonary tree of its secretions. Almost always an infant revealing such lung signs will be in a state of malnutrition, with anorexia, irritability, disturbed sleep, and sometimes persistent and harassing cough.

Under careful treatment, a few of these infants recover by aid of hygienic measures, coupled with heliotherapy. (See Meth-

ods.) Some therapeutists are advocates of the use of calcium and creosote, but on the whole little can be expected from any form of drug treatment. Cod-liver oil may be of value for its nutritive qualities and for its fat-soluble vitamine content.

Glandular tuberculosis, a common manifestation of the infection in older children, is unusual during infancy although it is met with increasing frequency after the twelfth month. The work of Gohn on the bronchial gland infections of children may be accepted as proof that tuberculosis of these glands is but a sequel of pulmonary involvement. It is rare that lung lesions heal and leave the glands themselves the chief concern. In certain unusual cases, which almost always occur during childhood rarely before the twentieth month, a persistent, aggravating cough, pallor, malaise, slight fever, and radiographic evidences of hilus infiltration will make the diagnosis of mediastinal tuberculous adenitis possible. Such a diagnosis should lead to the establishment of a regimen including heliotherapy and measures looking to improvement of nutrition.

Far more frequently the cervical glands are mistakenly pronounced to be tuberculous. It must not be forgotten that the posterior cervical glands drain the posterior pharynx and scalp, while the anterior cervicals drain the pharynx, fauces, tonsils and floor of the mouth. Only when the swelling of the glands persists through many months is there reason to suspect that they are tuberculous. Even then such a diagnosis must not be made until all chronic lesions of the scalp, nose, throat, teeth, and mucous membranes of the mouth have been canvassed as possible origins of an infection productive of the chronic adenitis.

The tuberculous gland throughout its long infiltrative stage is painless, discrete, moderately large, and smooth; it adheres neither to the skin above it, nor to the tissues beneath. Early in the disease, it is rare to have fever when tuberculosis is the etiological factor of the adenitis. Later a slight fever may be one of the manifestations, especially when secondary infections complicate the clinical picture; some matting of groups of glands may appear, and the skin may become adherent to them. Frequently caseation takes place in some of the glands, usually in only one or two. The adherent skin over these becomes red; if the process is allowed to continue, the skin breaks down and a caseous and grumous material evacuates itself, leaving a sinus which will remain indefinitely unless it is treated.

Chronic enlargements of the glands of the axillæ and of the groins, if they occur during infancy, are even less frequently of tuberculous origin than are enlarged cervical glands. When a generalized enlargement of the lymphatic glands becomes apparent, the spleen and liver should be carefully examined and a study of blood smears should be made at once, so that the onset of a blood or nutritional dyscrasia may not be overlooked.

The *treatment* of tuberculosis of the glands of the neck is the same as that for the other forms of tuberculosis. Attention to proper feeding, provision of ample fresh air, rest and heliotherapy are indicated. In addition, it is of advantage to use roentgenotherapy; ½ an erythema dose given over the swollen glands and repeated in 10 and 20 days is of distinct value in most cases. The ultra-violet ray, applied by the Germans and elaborated in this country by Alfred Hess, is said to be very efficacious. The formerly popular method of wide incision and extensive dissection should have no place in the therapeutics of the modern physician. The brilliant results that follow simple hygienic treatment of glandular tuberculosis attest its value. Even after the most skillfully applied hygienic therapeusis, resort must be made to surgical interference in a few cases; but surgical treatment will rarely be necessary. When a persistently discharging sinus has formed, the gland which lies at the bottom of the sinus must be excised.

Local applications of iodine and methyl salicylate are frequently used in the treatment of chronically inflamed nontuberculous glands. No rational therapeutist imagines that they can have any great influence on the local pathologic conditions except as they are absorbed into the circulation and reach the enlarged glands through the blood stream. It is possible that in this way they may hasten resolution and absorption. Hot compresses and poultices, or ice bags, while they may be of value in acute adenitis, have no proper place in the treatment of a chronic adenitis, whether it be tuberculous or nontuberculous.

The group of lymphatic glands most frequently affected with tuberculosis during the first two years of life is that lying in the mesentery and in the retroperitoneal spaces. An adenitis without peritonitis sometimes occurs, but it is unusual. Most often it is a combination of these inflammations which gives the clinical picture of **tuberculous peritonitis.** For purposes of consideration, tuberculous peritonitis may be divided into a dry form

and a form with effusion. In early childhood, a third form, the proliferative, occurs. In reality, this is but a variety of the dry type, although the pathologic changes that follow the extravasation and organization of an excessive plastic exudate produce a specific clinical picture.

In the *dry form* of the disease, the child presents all the nutritional disturbances inherent to tuberculosis of whatever nature, such as anorexia, loss of weight, slight fever and some mild abdominal disturbance. On examination of the abdomen, which is rarely very tender, definite tumor masses in characteristic situations may be palpable. These masses vary in size and in consistency, and they are usually accompanied by a palpable thickening of the peritoneum. The abdomen loses its soft, velvety feeling and acquires a harsh quality. In a majority of the cases of this type of peritonitis, palpation will reveal a firm sausage-shaped mass which begins on the left side of the abdomen at about the line of the umbilicus and runs downward to the right iliac fossa. The mass follows the course of the free border of the mesentery. Another tumor which is frequently encountered occurs in the right iliac fossa. A third lies above the umbilicus and to its right; and in many cases, other smaller, widely scattered masses representing enlarged glands are readily demonstrable. When the process is very acute and when the group of glands in the right iliac fossa is involved, some difficulty may be encountered in differentiating tuberculous adenitis from a subacute appendicitis.

In the *proliferative* type of the disease, the clinical picture is that of a moderately distended, rigid abdomen. Palpation of the belly reveals nothing to the examiner because of the deposition of thick layers of organized lymph which sometimes attain a thickness of 1 to 2 inches. In such a case, constipation is profound because the intestine is fixed as though it were set in a bed of mortar. Because of the intestinal fixation, abdominal pain is persistent and distressing. Sometimes the process extends along the spermatic cord and involves the epididymis, testicle and tunica vaginalis. The cord and the testicle become lost in a large, solid mass several times the size of the normal structures.

These dry forms of tuberculous peritonitis have a less favorable prognosis than *tuberculous peritonitis with effusion* into the peritoneal cavity. In the latter form of the disease, if the patient be seen in the early stages, masses of glands will be palpable in

the abdomen. These masses will have the same general distribution as those found in the dry variety of the disease, but they will rarely be so large. Later on the presence of shifting dullness will be evidence that fluid is accumulating in the peritoneal cavity, and within a week or ten days considerable quantities can be demonstrated by the ordinary methods of percussion and ballottement. In younger children, Koryani's sign of shifting dullness, disclosed by percussion over the sacrum, is a valuable means of confirming a suspicion that small amounts of fluid are present in the peritoneal cavity. In eliciting this sign, percussion is first made over the sacrum with the child in the erect position; the child is then inverted and held so that the pelvis is the highest part of the trunk, and percussion over the sacrum is repeated. If the note obtained in the first percussion is dull and that heard in the second is tympanitic, the presence of free fluid in the abdominal cavity may be assumed.

There has been much difference of opinion about the value of surgical intervention in tuberculous peritonitis. Surgeons have operated, and if it happened that the case was a favorable one, the patient recovered, and the result was taken as a confirmation of the opinion that surgical intervention was of value. On the other hand, the inadequacy of surgical procedure in unfavorable cases was overlooked in the discussion. There can be no doubt that it is entirely unjustifiable to operate on a case of the dry form of peritonitis unless it has become necessary to relieve an intestinal obstruction. In a large proportion of cases, incision will be followed by a chronic discharging fistula. The more experienced a surgeon is in the treatment of the dry tuberculous peritonitis of children, the more loath is he to enter the abdominal cavity. In the proliferative forms, it is perfectly useless to operate even in the presence of a definite obstruction, for no amount of surgical manipulation can relieve the interference with the function of the bowel.

It is not so easy to be dogmatic about the wisdom of operation when the peritoneal cavity is filled with effusion. There is no question that many children have recovered promptly after an abdominal incision with evacuation of the fluid. On the other hand, many have made quite as dramatic and complete recovery without surgical aid. Therapeusis based on hygienic and nutritional methods, especially on heliotherapy (see Methods), is entirely satisfactory except in that very small proportion of cases in which the accumulation of fluid is so great that its pres-

sure on the diaphragm interferes with the movements of the heart and lungs.

Apart from its relation as complication of involvement of the lungs, **pleuritis** has little place in the pathology of tuberculosis of infancy. Once in a decade perhaps, one may encounter a nonpurulent effusion into the pleura which is self-limited and goes on to complete recovery after the aspiration of the fluid.

Local treatment of **tuberculous synovitis** is ineffective, but the swellings may be favorably influenced by dietetic and hygienic measures directed toward aiding the resistance and increasing the nutrition of the infant.

In infancy, tuberculosis of the **kidney, bladder** and **testes** is almost unknown except as a part of a generalized tuberculosis. Occasionally tuberculosis of the penis results from ritualistic circumcision. See chapter on Genitourinary Diseases.

Keratitis occurring in the first two years of life is more frequently of syphilitic than of tuberculous origin. The differential diagnosis between the two conditions is made with extreme difficulty, although in many cases of syphilitic infiltration of the cornea, minute gummatous masses may be visible at the periphery of the cornea. There is no local *treatment.* Amelioration follows properly devised constitutional treatment.

Tuberculous **ulceration of the nose** and **of the nasopharynx** occurring at this age are always complications of a generalized tuberculosis. Tuberculosis of the **tonsil** does occur and may be followed by tuberculosis of the cervical glands. The lesions are usually discovered in the pathologic laboratory, on examination of the removed tonsil.

Epitome of the Treatment of Tuberculous Disease

Given a case of generalized tuberculosis, the *treatment* is essentially expectant. Anorexia interferes with the child's acceptance of food sufficient for its proper nourishment. The vomiting, which is a distinctive feature, is not amenable to any form of drug therapy, and the progressing malaise responds neither to tonics nor to stimulants. The convulsions present at the onset of the terminal stage in which meningitis is the striking feature can be diminished in number and in intensity by lumbar punctures; the headache, even though it be excruciating, will be relieved for a time by the same procedure. Against the lethargy, somnolence and final coma medicine is powerless.

The management of the few cases of infantile tuberculosis with limited and localized lesions is essentially a matter of regimen. Abundance of fresh air, cleanliness, massage to improve the circulation, dietetic therapeutics and heliotherapy are included in the necessary treatment of such patients.

The former view that overfeeding was an essential in the dietetic treatment of tuberculosis is no longer held. A properly devised diet for a patient with tuberculosis will call for food intake about 20 per cent in excess of that needed to meet the energy requirements of a normal child of the same age and weight. This excess is needed because such children are usually thin, lose heat rapidly, and suffer from fever with an accompanying waste of tissue. A liberal fat ration is essential for them, but the feeding of great quantities of cream and egg yolk is to be deprecated. Milk is a fundamental food for these patients and most dietaries call for about one quart daily. It is well to remember that children at this age may suffer from metabolic disturbances, apparently the result of an intestinal intoxication which follows the ingestion of large quantities of milk. It is a simple matter to protect these cases by the addition of liberal quantities of lactose or dextrin to the diet. At least 1½ ounces of either one or the other should be added to each pint of milk in the dietary. These carbohydrates are of great value because they are easily digestible, readily available, and provide the body with energy. They are of further advantage in that the excretion of the end products of their metabolism entails no strain on the kidneys. They are also useful in maintaining the glycogen reserve at a high level and there is some reason to believe that an adequate glycogen reserve is one very important factor in the maintenance of the resistance to invading bacteria, including the bacillus of tuberculosis.

Butter is a valuable foodstuff in the treatment of tuberculous babies, and it is much better digested than an equivalent amount of cream. Many babies of from 18 to 24 months will take 1 or 2 ounces of butter a day with no disturbances of digestion. Fruit and vegetables, because of their salts are to be used freely. Meat, in the form of scraped meat patties, should be a part of one daily meal. However, it must not be forgotten that tapeworm eggs sometimes lurk in the best appearing beef. One or 2 egg yolks daily may be given to a child who has passed its sixteenth month. Before this age, 1 yolk is usually all that will be well tolerated. The yolk may be given raw, added to the milk, al-

though many children are unable to digest it in this form. For such, there are other methods of preparation which will improve the digestibility of the egg-fat. For instance, the yolk may be beaten into very hot fruit sauce or into boiling broth; it may be worked up with flour and the mixture be added to boiling broth, and cooked for 20 minutes; or an egg may be boiled for 30 minutes and the hard yolk creamed with butter. This may then be added to vegetable or fruit pulp or to broth, or it may be used as a sandwich filling. Bread, toast, crackers, zwieback are all to be given freely. Malted milk and ovaltine are of value if they are relished, but urging the child to take such foods to the point of satiation of its appetite is to be condemned.

The feeding schedule best adapted for most tuberculous children in the second year calls for 4 meals a day, but when anorexia is a feature, it is better to give only 3. In such a case, the sandwich or fruit lunch between meals is detrimental. The details of the number and size of the meals must remain a matter for decision in each individual case.

Certain clinicians believe that calcium added to the food is of great value in cases of tuberculosis. No harm can come from the addition of calcium carbonate in 5 grain doses to 4 feedings a day. Because these children are nearly all anemic, it is a good plan to combine the calcium with 2½ grains of the saccharated carbonate of iron, and when much starch is being given in the attempt to provide a high calorie diet, 2½ grains of taka-diastase will often prove to be a valuable aid to digestion. These three substances may be combined in the same powder.

Cod-liver oil may be given; it often proves of use as a food, and as a stimulant to the appetite and to nutrition. Some children tolerate it badly but most will accept it if it is given in a 33 per cent emulsion. Simple elixir is a good flavoring agent to disguise effectively the unpleasant taste of the oil.

Heliotherapy, as devised and practiced by Rollier, has brought a really potent weapon to us in the struggle against the subacute and chronic forms of tuberculosis. Practicing in Switzerland, at a high altitude, Rollier has shown that it is possible to expose naked children to the direct rays of the sun even in winter weather, provided they have been trained to tolerate the lowered temperature by gradual inurement. The sun's rays must be allowed to fall directly on the child's unclothed skin without the intervention of glass, fabric curtains or any other interrupting me-

dium. Time must be taken in training the body to tolerate the actinic rays. It is well to begin with an exposure of 3 or 4 minutes of a limited area of the body, perhaps not more than 8 or 10 square inches. The site chosen for the first exposure is usually the location of the disease—the abdomen in tuberculous peritonitis, the spine in Pott's disease, the hip joint or knee joint if these be affected. Daily a larger area is exposed and 2 or 3 minutes longer given for the exposure until finally the whole body is brought under the direct influence of the sun and left exposed from 2 to 4 hours daily. When the time comes that the child is able to take a full exposure, provided he is able to be about, it is well to provide him with a very short-sleeved, short-legged and exceedingly low-cut bathing suit which is the only garment he is to wear during play hours. This method of heliotherapy is valuable not only in the treatment of tuberculosis, but also as an aid to dietetic and hygienic measures in many forms of malnutrition as well as in other chronic diseases.

General massage is a valuable auxiliary in the treatment of tuberculosis because it improves the circulation, stimulates nutrition and increases muscle tone. However, it is impossible to overemphasize a protest against the too common practice of certain mechanical therapeutists who vigorously manipulate and violently massage areas in which the tuberculosis process is acute.

Diphtheria

It is fortunate that diphtheria is rarely encountered during the first 2 years of life. However, it is at this period that one of the most dangerous forms, which is at the same time one of the most difficult to diagnose, appears. This is **diphtheria of the larynx,** the "membranous croup" of our forefathers. **Nasal diphtheria** is a form of the disease which is sometimes met with and often overlooked at this age.

The diagnosis of **diphtheria of the pharynx** rests on the same criteria that guide us when older children are the victims. The slight rigor at the onset, the fetor, the presence of a white membrane which makes its appearance first either as a slight patch coming down along the posterior pillars of the pharynx or as discrete patches arising from the crypts of the tonsils, all these are significant. These patches on the tonsillar crypts later coalesce to form a continuous membrane not very readily detachable from the mucous membrane; detachment is followed by characteristic bleed-

ing. This membrane is rather felty in texture, is thick and is quite unlike the pellucid, whitish membrane of a streptococcic or a pneumococcic affection. Young children with pharyngeal diphtheria very often have enlargements of the anterior cervical glands, especially those in the upper part of the anterior triangle. These enlargements have sometimes been mistaken for the swellings of mumps. A proper examination of the throat should exclude such an error. Cultures on blood agar will settle the diagnosis within 12 hours, and a skillful pathologist can very often determine the presence of Klebs-Loeffler bacilli in the smear and thus save 12 hours delay in initiating treatment. When the clinical diagnosis seems reasonably certain, antitoxin injections should be used without waiting for laboratory confirmation.

Nasal diphtheria may be either acute or chronic. The **acute form** is really a diphtheritic involvement of the superior surface of the soft palate and is accompanied by nasal obstruction and characteristic fetor. Examinations of the throat will show a fine line of membrane along the posterior edge of the soft palate, and a culture of the pharynx, nasopharynx or anterior nares will reveal the pathognomonic organism. These manifestations may mark the first stage of what later becomes a pharyngeal diphtheria. This type of nasal diphtheria is accompanied by marked constitutional symptoms—fever and malaise—and is the most frequent forerunner of palatal paralysis.

The **chronic** or **carrier type** of nasal diphtheria, while not at all uncommon in children of school age, is more frequent than the acute type of nasal diphtheria during the second year of life. The child will have an excoriation on one side of the lip; there will be a blood-stained nasal discharge; and examination of the anterior nares will reveal larger or smaller patches of membrane from which diphtheria bacilli may be cultivated. Constitutional symptoms rarely appear in this chronic type of diphtheria of the nose, and subsequent paralysis is unusual.

Laryngeal diphtheria may occur as a complication of the pharyngeal involvement or as the only manifestation of a diphtheritic infection. As a rule, the constitutional symptoms early in the disease are not so severe as in the other types and they sometimes fail of recognition at this time. Later, when the membrane has interfered with air entry and the signs of pulmonary embarrassment develop, the effects of the specific toxin of the disease are added to increase the gravity of the picture. The brassy, inces-

sant, paroxysmal cough of diphtherial laryngitis, once heard, can never be mistaken.

It is wise to make an immediate culture on blood-serum and to study a smear of the secretion taken from the throat in every case termed "croup" by the parents, especially where the brassy cough is present and persistent and any cyanosis is manifest. Stained with new-blue, the diphtheria bacillus, if present, will be seen in Chinese-letter-like groups. The bacilli are of moderate length and, with this stain, their polar granules show distinctly as purple dots against a background of blue-stained protoplasm.

The efficacy of antitoxin, when used early, is so great that no chance of delay should be allowed. If laboratory facilities are not available, an immediate intravenous or intramuscular injection of 15,000 units of diphtheria antitoxin should be given on the clinical signs alone, especially if there is no remission of the cough and if cyanosis, forced inspiration and stridor appear. Should the case be one of a streptococcus or pneumococcus laryngitis with croup, no harm will be done; while if it be diphtheria, the child's life may depend on the early and sufficient use of antitoxin. Within 24 hours after the use of initial intravenous or intramuscular injection of 15,000 units of antitoxin, the membrane should loosen and be expelled. It is a very common thing, however, for a second membrane to form, and this is often the source of much trouble. Unless the first injection was intravenous, it is good practice to give a second injection of 15,000 units 24 hours after the first dose; this method undoubtedly saves many cases of laryngeal diphtheria from the need of intubation. (See page 430.)

When the physician has been called late in the course of the disease, or when the antitoxin given was insufficient, intubation or tracheotomy will be needed. The detailed instruction for passing an intubation tube is found in the chapter on Methods. It is an especially delicate operation in infants and should be undertaken only by an expert. The slightest undue force may produce a fatal injury. In the absence of a practiced intubator, tracheotomy is an operation possible to any reasonably trained surgeon and is the method of choice.

Inhalations of plain steam or steam impregnated with such drugs as creosote, phenol, benzoin, oils of eucalyptus and pine seem to aid in the relief of the dyspnea and stridor. Inhalations of calomel vapor are sometimes of service. Emetics are dangerous, but the writers have seen the expulsion of a large cast

that was blocking a tracheotomy tube follow the use of apomorphine; it was possible, after the cast came away to replace the intubation tube, which then remained clear and allowed the child to breathe without further difficulty. No other method of local treatment can be of any great value, and the removal of the laryngeal exudate must wait on the specific action of the antitoxin.

Diphtheria occasionally attacks the **conjunctiva.** It is more likely to arise from a direct infection rather than from extension through the lachrymal duct. It is a severe complication at best, even with antitoxin as a weapon, and it may cost the patient the sight of the eye. The *treatment* is essentially the same as for a pharyngeal diphtheria. In addition, frequent irrigations of the eye with warm alkaline solutions are needed in order to keep the discharges from accumulating.

Diphtheria may attack the mucous membranes of the **vagina** and **vulva,** but this distribution is most rare during infancy.

The *treatment* of diphtheria with simple pharyngeal involvement calls for the immediate intravenous or intramuscular injection of 8,000 or 10,000 units of antitoxin. When the intramuscular route is used, a repetition of the same dose within 24 hours is indicated, except in the mildest cases; in these, a single injection suffices. W. H. Park has shown that the single large dose of antitoxin, given by intramuscular or intravenous route, on an early day of the disease is more efficient than 12 hour repetitions of smaller doses. It is better to err on the side of safety and give the initial large dose, repeating it once or twice according to the clinical indications. One lesson that is clearly brought out by the work of Park and of other experimenters in this field is that any dose of antitoxin, no matter how large, given late in the disease, cannot undo the damage already done by the diphtheria toxin, when treatment has been unduly delayed.

The physician has constantly to meet the protesting parents' fear that antitoxin of itself is harmful, and that heart failure and palsies are the result of its use. If we would but turn to the clinical papers of Baumler, Senator and Trousseau and a dozen others who dealt with this disease before the days of antitoxin, we would find ample refutation of this erroneous conception. These writings are replete with case records showing that amongst patients who survived the toxic stage of diphtheria, late heart failure and paralyses were commoner than they are today.

Every physician has an uncomfortable feeling when he injects heterologous serums into human beings, for he has ever before his mind the unhappy instances of anaphylactic deaths which have occurred at rare intervals. However, even this remote contingency need cause little alarm at the present time since we have learned that a previous desensitizing dose of serum will protect almost any sensitive person. In order to be on the side of safety, it is well never to inject a massive dose of antitoxin at once. A preliminary subcutaneous injection of ½ c.c. to 1 c.c. of the serum to be used should be made, and the intravenous or intramuscular injection of the remainder be delayed for from 30 minutes to 1 hour. If there is any reason to suspect that the patient is hypersensitive, a second preliminary dose of 1 c.c. of the serum should be injected into a muscle; this injection should precede the intravenous by an hour.

The superiority of the vein as a route for the injection of antitoxin is no longer a matter for discussion. In 1904, one of the writers began to use the intravenous route in the San Francisco Hospital and he has used it ever since with gratifying results. There is no doubt that in exceedingly toxic cases and in patients almost moribund there is the advantage of prompt action that can be had in no other way. It is advantageous that the serum for intravenous injection be well diluted with normal saline solution, and it is better if at least 15 minutes can be taken to complete the operation; otherwise, profound shock may ensue. Ordinarily, intramuscular injection is quite satisfactory and it is a simple thing to do. Although it is followed by some muscular tenderness, this distress is very little more than follows an injection into the subcutaneous tissues, while the action of the antitoxin is much more rapid and effective. Care must be taken not to allow even a few drops of the serum to infiltrate the skin, for a painful or even a sloughing area may be produced.

In administering antitoxin subcutaneously, some authorities recommend the back as a site for injection. As there is always tenderness following such an injection, it is unwise to use this area, for it prevents the child's lying comfortably in bed and tends also to aggravate restlessness and to produce sleeplessness. If the subcutaneous route is decided upon, the loose tissues of the anterior abdominal wall are to be preferred. The globulin type of antitoxin should always be used because of its greater concentration. When it is used in older infants, it may be in-

jected into the triceps muscle; there is little pain following injections into this area as it is less sensitive than other available sites and the chance of infection is less than if the buttocks are used. Once the fontanel is closed, intravenous injection is difficult, but if it is found necessary to enter a vein of an older infant, the external jugular will almost always be available. In young children with open fontanels, the longitudinal sinus is readily accessible.

As an adjunct to the use of antitoxin, *local treatment* is valuable in the pharyngeal and nasal types of the disease. An irrigation of the pharynx with a hot 2 per cent sodium bicarbonate solution is grateful and cleansing. The same solution may be used to irrigate the nose in acute nasal involvement. In the chronic nasal type of diphtheria, the application of silver nitrate to the areas from which the membranes are shed will tend to prevent their persistent recurrence. A time-honored but none the less valuable adjunct to the use of antitoxin is the prescription of the tincture of ferric chlorid and glycerin. There is no question but that the use of this formula often improves the general condition of the patient and has some local action which makes the pharynx more comfortable.

Treatment of the patient who has been cured of his clinical diphtheria but who still carries Klebs-Loeffler bacilli in his nose or throat is one of the most perplexing problems with which we have to deal. The concensus of opinion seems to be that one of the few effective means of reaching this *carrier* problem is to remove the patient's tonsils. This operation is advisable although it can have little effect when the nose or nasopharynx are harbors for the organism. Irrigations have been tried, and insufflations of various powders (acetanilid, bismuth formic iodid and aristol) are recommended. Mercurial oil is the most effective of local treatments; this should be instilled into the nostrils and swabbed into the nasopharynx and pharynx 2 or 3 times daily. Such treatment will usually succeed in rendering the patient germ-free, although in some instances only after a lapse of 6 or 8 weeks. French clinicians report that insufflations of dried powdered antitoxin are sometimes effective.

The **postdiphtheritic palsies**, as they occur in infants, may be divided into two classes: those which occur early during the course of the disease; and those which are tardy, coming on 4 to 7 weeks after the last acute symptom of the bacterial invasion

has disappeared. Into the first category fall palsy of the palate and weakness of the ocular muscles. These palsies are self-limited and pass off promptly as the antitoxin becomes effective. The diaphragm may be involved in these palsies, and the subsequent respiratory failure may become the cause of death. Continuous artificial respiration for 24 or 48 hours has been used in the treatment of the condition; one patient of Marriott's was saved by this procedure.

In later palsies, the palate, the pharyngeal constrictors and the extremities, especially the lower, one or all may be involved. When the eye is damaged, the power of accommodation is lost; this does not interfere with the baby's comfort and it is likely to be overlooked. The palsies of the palate and pharyngeal constrictors interfere with swallowing and may lead to a fatal issue. Paralysis of the palate is alarming to the parents as the child chokes and sputters and regurgitates fluid through the nose when it attempts to swallow. Damage to the lower extremities interferes with progression and keeps the child bed-ridden. All of these palsies are self-limited and recover without any treatment; rest and the provision of good hygienic surroundings are indicated.

At the same time that the early palsies occur, rapid and *acute degenerations of the heart muscle* may take place. Slowing of the pulse is extreme, and the children so affected may die very suddenly within a few hours after the first appearance of the cardiac symptoms. The heart lesions, however, are more frequently delayed and appear in the later stages of convalescence or some time afterward, along with the late palsies. Bolton's study of these early cases has shown that not only is the heart muscle itself subject to degenerative changes, but the neurons that originate in the vagus nerve are also affected. The possibility of the sudden development of cardic failure in a diphtheria patient who is apparently doing well makes it incumbent on the physician to exercise the utmost precaution. Great care should be observed that no undue strain is put on the cardiac apparatus. Constipation and consequent straining at stool are a frequently overlooked cause of cardiac stress in the course of diphtheria. Careful attention to the condition of the bowels is therefore a very important matter in the treatment of those patients who show cardiac weakness. The child should be kept in bed even when the disease seems mild and the progress satisfactory. Five weeks

is not too long for even the slighter cases to remain recumbent. In the more severe cases, the limit of the stay in bed can be decided only by the physician in attendance, after a careful study of the cardiac response to effort.

In the early oncoming form of cardiac degeneration, camphor in oil is of great value and it should be given by hypodermic injection at 2 to 4 hour intervals. When the pulse is slow, 1/400 grain of atropin for a 25-pound child, repeated once daily is often of use. The most valuable of all drugs under these circumstances is opium. Cardiac patients are very restless, and their restlessness and continued movement throw a persistent strain on the damaged heart. When this restlessness is urgent, it is wise to give enough opium (codein or morphin) to keep them drowsy and semistuporous for several days, until the toxemia is diminished and the heart has had opportunity to undergo some regeneration. Sleep is the greatest restorer in this as in other acute exhausting diseases, and opium has the further advantage of inducing sleep.

Brandy has an ancient and apparently a well deserved reputation as a drug in the treatment of cardiac affection in diphtheria. Given in 10 to 15 drop doses every 3 or 4 hours, the drug is of advantage.

Food should be chosen for its ready digestibility. Large quantities of fluid are contraindicated because of the strain they may throw on the circulation. Sugars, in reasonable dilutions, are of great value. Other carbohydrates in the form of the finer cereals may be used. Butter fat and fruits will also find a place in the dietary. The chief reliance should be placed upon broths thickened with such carbohydrates as sago, rice, barley or farina. The preserved and stewed fruits are used because of the sugar they carry; and the simple pastes (vermicelli and pastine), with butter and salt, are valuable. The milk intake can well be limited to a pint a day and this can be reinforced with 1 or 2 ounces of dextrose or lactose.

The *Schick reaction* has come into use in the last few years as a means of determining the presence or absence of a natural immunity to diphtheria. The medium used for the test is a diphtheria toxin and the amount injected is equivalent to $\frac{1}{50}$ of the dose that would kill a 250 gram guinea pig. The injection is made into the layers of the skin of the flexor surface of the forearm. A positive reaction represents the absence of circulating anti-

bodies and the evidence of such reaction is the appearance of a papule of ⅓ to 1 inch in diameter at the site of injection. The true Schick reaction takes about 48 hours to develop, and the skin at the site of injection is characteristically indurated; but there are certain pseudoreactions of an anaphylactic or irritative nature which are less circumscribed, less indurated and a lighter red in color. These may appear at any time after the injection, and care must be taken not to mistake them for a true Schick reaction. If this reaction occurs in an exposed individual, it indicates a lack of natural immunity and the individual should receive a prophylactic dose of 1,000 units of antitoxin.

As an immunizing agent against diphtheria, the toxin-antitoxin mixture has had an extensive trial by Park and his coworkers in the New York Board of Health. They have found the procedure to be absolutely harmless and they recommend it, because in their hands 3 injections of the toxin-antitoxin mixture given at weekly intervals have achieved a passive immunity in 97 per cent of susceptible cases. In these cases, susceptibility was determined by positive Schick reactions. Full immunity does not appear until the tenth week, although there is reason to believe that acquisition of immunity begins immediately after the first injection. From a series of cases observed for more than 3 years, it is known that immunity produced in this way can last for that length of time, and it is believed that it may be permanent. In older children and in adults, the injection is sometimes followed by a severe local and general reaction. This accident seldom occurs in infants, although it is not advisable to use the method before a baby is 6 months old.

Some authorities, among them even so conservative an organization as the United Public Health Service, advise that the Schick procedure be practiced on all children between the ages of 6 months and 5 years and that the toxin-antitoxin protection be given to all who show a low degree of natural immunity to diphtheria.

It has been demonstrated that natural immunity to diphtheria is based upon the presence of antitoxin in the blood of the immune individual. Complete immunity is insured by the presence of at least $\frac{1}{30}$ of a unit of antitoxin in each c.c. of the blood. Those who possess less than $\frac{1}{30}$ unit of antitoxin, and who there-

fore lack immunity, react in a very definite way to injection of diphtheria toxin.

The Schick test has determined that susceptibility varies greatly at different ages, as is shown in the following table:

	Susceptible
under 3 months	15%
3 - 6 months	30%
6 mos. - 1 yr.	60%
1 yr. - 2 yrs.	70%
2 yrs. - 3 yrs.	60%
3 yrs. - 5 yrs.	40%
5 yrs. - 10 yrs.	30%
10 yrs. - 20 yrs.	20%
over 20	12%

Some recently reported results of Park and his coworkers illustrate very vividly the advantages of the early administration of antitoxin and as well bear testimony to the superiority of the intravenous route for its administration. A reference to the following table will show that 1000 units of antitoxin given intravenously is equivalent to 20,000 units given subcutaneously.

Relative Efficiency of Antitoxin with Different Modes of Administration

Age in years	Number units	Mode of administration	Schick 20 hours previously	Schick 6 hours previously	Schick 4 hours previously	Schick 2 hours previously	Schick simultaneously
6	1,000	subcutaneous		+++	+++	++	+−
4	1,000	intramuscular		+++	++	+−	
5	1,000	intravenous		+	+−		
5	20,000	subcutaneous		+−	+−		
6	10,000	intravenous	++				
6	10,000	intramuscular	+++				
6	20,000	subcutaneous	+++				

Measles

Measles is essentially an affection of the school age and of run-about childhood. It is fortunately rare in the first year and not at all frequent in the second. When it does occur in children at this period, there is usually a very clear history of contact with an older patient. Many papular and erythematous rashes are classified by parents as measles; for this reason the statement may be made that the patient has had measles 3 or 4 times. Unless there has been a house epidemic, such diagnoses may be dismissed as improbable. History of measles without known contact should lead us to investigate the possibility that the eruption is a cutaneous expression of syphilis.

The earliest signs of the disease are malaise and a marked injection at the inner canthus of the eye, followed by fever. In the mouth, the earliest indication is a general reddening, which appears on the soft palate and pharynx, together with a very superficial erosion of the buccal epidermis. There is injection of the gums, and on the second or third day of the malaise the macular eruption first described by Koplik appears on the *buccal mucous membranes.* Toward the end of the third or fourth day, the characteristic rash comes out on the skin, usually at first behind the ears, whence it spreads over the entire cutaneous surface.

From the beginning, cough is striking and unpleasant; at first it is an unproductive effort, incessant and aggravating. Rhinitis with sneezing and conjunctivitis with lachrymation and photophobia are present in most cases from the onset. The face is swollen and the eyelids thickened; very often the conjunctiva secretes a purulent discharge which causes gumming of the blepharal margins. There may be prodromal hyperpyrexia of some days' duration. Cases are on record in which the fever lasted for 2 weeks before the appearance of an eruption explained its cause. On the other hand, as a prodrome there may be subnormal temperature, although this is unusual. At times, the fever and the toxemia are so extreme at the onset of measles that marked cerebral symptoms with convulsions or meningism may be part of the picture. Comby has proved by lumbar puncture and cytologic observations that during measles the meninges are involved in a manner analogous to the skin, so that such profound central nervous symptoms are readily understandable.

Following the prodromal symptoms and the first appearance of

the eruption behind the ears and on the face, the rash spreads over the body until in 2 days it has covered the entire skin. At first, the eruption consists of discrete macules which are somewhat scattered. Each day more macules appear and the reddening of the skin becomes more intense until by the third day after its onset the eruption is generalized. In severe cases, the rash is confluent, while in the milder cases the skin manifestations remain macular and sparse. On the third or fourth day following the onset, fading begins; 3 or 4 days later, slight staining of the skin with slight desquamation is all that remains.

During the stage of eruption, all of the superficial lymphatic glands are enlarged. The occipital group is rarely involved to the same degree that it is in German measles, and this fact provides one of the most important differential points between the two diseases. A characteristic but not inevitable feature of the fever in measles is the remission of temperature that often occurs on the second or third day of the prodromal stage. A few mild cases run an afebrile course. In most instances, however, with the appearance of the rash, there is a continued high temperature without much variation, the fever lasting until the eruption begins to fade. Hyperpyrexia is a feature of the more severe cases. If the fever continues during the period of convalescence, complications must be suspected and sought.

The *complications* are predominantly those of the ear and respiratory tract. A mild bronchitis is really a part of the clinical picture of the disease. Severe bronchitis and bronchopneumonia are common complications. The bronchitis may persist and become subacute or chronic, remaining long after the symptoms of the measles have disappeared. Some cases with this involvement develop tuberculosis and die after some weeks or months. Lobar pneumonia may occur as an accidental complication. Streptococcus or pneumococcus infection of the larynx is a development greatly to be feared during an attack of measles in infancy. A pseudomembrane may form and present the same obstructive symptoms that are found in diphtheritic laryngitis, but specific antitoxin is wanting and treatment is much less effective than in diphtheria. As a further complication of such laryngitis, ulceration of the larynx, with edema and subsequent scarring, may take place. In the course of these affections, a sudden acute edema of the larynx may supervene and cause instant death. Double infections with measles and diphtheria are not unknown;

when they occur, they are almost uniformly fatal. The early and sufficient intravenous use of antitoxin is urgently indicated.

Middle ear inflammation caused by extension from the inflamed throat through the Eustachian tube occurs in a large number of cases of measles. It may be mild or severe; it is usually self-limited, although the mastoid cells sometimes become involved. In the course of measles, it is imperative to examine the ear frequently in order that a suppurative otitis media may not be overlooked.

The desquamation of the epithelium in the throat and mouth sometimes results in a tenderness that is distressing and prevents the child from taking any but the most bland foods. This condition also contributes to the intractability of the cough. The same kind of damage affecting the epithelium of the nose gives rise to a thick, purulent discharge that accumulates and prevents free breathing, much to the infant's distress.

About 10 per cent of all cases of measles show a severe and intractable diarrhea. The stools are purulent and sometimes bloody. There is a good deal of pain in the belly accompanying this complication, but like most of the other complications of measles, it is self-limited.

In order to prevent the appearance of the disease among infants, it is necessary to use only reasonable precautions. Older children should be kept from close contact, and no one who coughs should be allowed admission into the same room with the infant. It is not probable that indirect contact is responsible for the spread of the infection. The virus is exceedingly vulnerable and there is little chance that it can be carried by clothing, books or toys. It is to be remembered that during the prodromal stage the victim of measles is most dangerous to those about him. Epidemics are largely maintained by contact with patients who have not yet begun to cough or to show any but the prodromal signs of the disease. It is probable that very little infection takes place after the acute coryza and conjunctivitis have subsided, even in those patients who develop chronic bronchitis.

The *treatment* of the uncomplicated case of measles is simply a treatment of symptoms. It should include attempts to control fever, when this is excessive; to relieve the distressing cough, to protect the photophobic eye from irritation; and to ease the itching of the skin, which sometimes is almost intolerable. It is necessary to combat a superstition, which is prevalent in the lay

mind, that the "striking in" of the measles is certain death. This belief leads to the use of measures that makes the patient exceedingly uncomfortable; such measures include insufficient ventilation, the use of excessive amounts of clothing and bed coverings, and the withholding of baths or even of spongings.

Pyrexia can be as effectively and harmlessly controlled by the use of hydrotherapy in measles as in any other febrile disease. A warm bath given twice a day calms the nervous symptoms, reduces the fever and makes for the comfort of the patient. When the skin is itching, the addition of ½ dram of cresol to 5 gallons of water for the bath will mitigate the suffering. After the patient is removed from the bath, a thorough dusting with powder—stearate of zinc or rice powder—will further aid to relieve the cutaneous irritability. A ½ per cent phenol ointment is also efficacious for this purpose if applied to limited areas.

The eyes are to be protected by darkening the room, but there is no need for the Stygian darkness so often enforced by anxious relatives. The conjunctivae can be treated by instillations of mild alkaline lotions. Alkalies are cleansing and more soothing than acids, and 1 per cent borax is to be preferred to a solution of boric acid. When the conjunctivitis is extreme, the use of a ½ per cent yellow oxide of mercury salve applied to the edges of the lids is followed by gratifying amelioration.

For the cough, nothing is more effective than the use of 5 minims of the syrup of ferrous iodide and 30 minims of the syrup of hydriodic acid to the dose, given with a few drops of glycerin in chloroform water as a vehicle. The purpose of the mixture is to increase secretion and in this way to relieve the irritating, unproductive cough. For 1 or 2 nights, it may be necessary to give $\frac{1}{16}$ to $\frac{1}{20}$ grain of codein or $\frac{1}{60}$ grain of heroin in aqueous solution to a 20-pound child. These drugs will effectively check the cough and promote sleep. It is unwise to add opium to cough mixtures for children of this age; this drug should be prescribed alone, and it should be retained for use in a single dose to be repeated not more than once or twice a day for a few days.

In those cases in which the rhinitis is profuse and purulent, an irrigation of the nose with warm borax solution is helpful. Care must be taken during the irrigation that the child is held erect with the head bent forward, so that the fluid is not forced into the Eustachian tubes (see Methods).

Steam inhalations given from a croup kettle are of advantage to those children whose cough is in the dry stage as well as to those who have laryngeal and nasal complications. Volatile oils added to the water may have some psychologic effect on the parents, but they have little practical advantage. (See Methods.)

The use of the iodides, if begun early, may prevent severe laryngeal complications. If a nondiphtheretic laryngitis should appear, inhalations of calomel vapor are often effective in checking the process. Rapid vaporization may be effected by placing 10 grains of calomel on a heated tin plate which is held by tongs. The vapor is allowed to enter a small sheet tent arranged over the child who is kept within this partially closed tent for 5 minutes while inhaling the vapor of the drug. The procedure causes a good deal of coughing and the ejection of tenacious secretions and pseudomembrane. One inhalation, possibly followed by another in 12 hours, often proves valuable in combating this complication.

The use of emetics is sometimes indicated when the larynx is involved. Of these drugs, apomorphine ($\frac{1}{60}$ of a grain as a maximum dose) is the most effective and has the advantage that the nausea it produces rapidly disappears. The wine of ipecac is also effective, but its action is not so prompt, neither is the nausea so quickly terminated. In very severe cases of laryngeal complication with a pseudomembrane and respiratory obstruction, intubation or tracheotomy may be needed. When these operations become necessary, antitoxin should be given intravenously, even without the identification of the Klebs-Loeffler bacillus.

A mild, bland diet should be chosen. During the early part of the disease, anorexia will interfere with the ingestion of food, but the child should be given an abundance of water. The addition of fruit juice to the water is sometimes gratifying to the patient, although when the mucous membrane of the mouth and throat is much denuded, fruits may cause distress. High calorie orangeade (see Chapter on Recipes) is excellent in that it meets both the nutritional and water needs at this stage. As the appetite returns, cereals, fruit sauces, junkets, puddings, jellies, toast, zwieback and toasted crackers with butter may be used to meet the nutritional requirements without unduly taxing the digestive processes.

Rubella (German Measles)

Rubella is a mild epidemic disease of rather frequent occurrence in the United States. Only on the rarest occasion does it appear in a severe form. Most often, the patient exhibits a rash which consists of discrete, pinkish macules scattered over the body; these appear first on the face, and later on the trunk and extremities. It is characteristic of the disease that the rash never occupies the entire body at the same time. At first, when it is found on the face, the trunk is clear; later, when it appears on the chest, abdomen and limbs, the face is unaffected. There is a mild reddening of the throat, and macules appear on the soft palate and on the buccal mucous membranes simultaneously with the appearance of the eruption on the face.

Prodromal symptoms are the exception, although in about one-fourth of the cases there is a day or two of slight fever. In some of these cases, there is a prodromal rash quite different from the later characteristic rubella eruption. When this appears as a transitory pinkish, general blush, it is difficult to differentiate from a mild scarlatinal rash. However, it rapidly disappears and is replaced within 24 hours by a macular eruption typical of rubella. The rash may disappear within 48 hours; rarely, it lasts for 4 or 5 days. The fading eruption of rubella is followed by a slight brownish staining of the skin, and infrequently by a mild desquamation. The occipital glands are always definitely swollen in a case of rubella, and this fact is an aid to diagnosis in doubtful cases.

No *treatment* is needed for these cases beyond the maintenance of cleanliness and hygiene. Isolation of the patient is essential in order to prevent the spread of the disease.

Parotitis (Mumps)

Epidemic **parotitis** is seldom seen in the first year and it is rare in the second year of life. When the disease does make its appearance in an infant, it will always be as a part of a house epidemic or it will have resulted from special contact with a known case. In all but few instances, the disease is trivial and it passes away after a short period of malaise, slight fever (although the whole course may be afebrile), and swelling of the parotid and other salivary glands. It is not unusual to encounter cases in which there is swelling of lymphatic glands about the angles of

the jaw and ears which is thought to be mumps; when the parotid is actually involved, there is a pathognomonic swelling in the area of that gland. This swelling raises the pinna of the ear away from the face in a manner no other swelling of this region achieves. At the same time, an examination of the buccal surface will reveal a swelling of Stenson's duct and a thickening and eversion of the mucous membrane at its opening. The lining of the duct swells more than the sheath, with the result that the swollen duct lining protrudes onto the membrane of the cheeks and appears as a small bright-red papule.

The only affection that can readily be confused is a **septic** parotitis in which the swelling is of much the same character. But in this disturbance, a drop or two of pus can usually be forced out of the mouth of Stenson's duct. The presence of some other source of infection, such as a suppurating appendix or an abscess, will be demonstrable. The blood picture too aids in the diagnosis. In a septic parotitis, there will be a polymorphonuclear leucocytosis; while in a case of mumps there will be a normal leucocyte count or a leucopenia with a preponderance of lymphocytes.

With the subsidence of the symptoms in one parotid gland, a similar swelling appears in the other, and sooner or later the submaxillary gland and occasionally the sublingual glands become affected. When, as sometimes happens, the parotids escape and only the sublingual or submaxillary glands are involved, the peculiar distribution of the swelling may render a diagnosis exceedingly difficult. In appearance, the swelling may be such as to mimic a Ludwig's angina but there will be none of the pain or depressive toxemia that accompanies the streptococcic infection of the tissues of the neck, nor will palpation reveal the brawny, indurated tissues that are constant in Ludwig's angina.

Mumps may be complicated by a *streptococcus* or a *staphylococcus* infection and there may be a concurrent cervical adenitis which may present a perplexing problem of diagnosis. Such a concomitant infection is sometimes the source of those rare cases of nephritis which appear during or after a case of mumps. A purulent otitis media may also follow from the same cause. Permanent deafness and eye changes which have been recorded are probably always very rare sequelæ of a complicating meningoencephalitis.

The fever in mumps rarely runs over 103° or 104° and very

often there is no fever after the appearance of the swelling. Pain is seldom severe, although sometimes a great deal of discomfort results from movements of the jaws, and some difficulty may be experienced in opening the mouth.

One of the rare complications of mumps in male infants is *orchitis*. In the course of a parotitis, the parenchyma of the testicle swells while the epididymis is but little affected. The pain induced by this complication is extreme. It is a matter for discussion whether or not girl children suffer from an analogous ovarian complication.

Acute *pancreatitis* with tremendous shock, excruciating pain and copious bowel hemorrhage has occurred; it was seen once in the writers' series of cases. The patient was a male and underwent two exacerbations of the abdominal symptoms and finally made a complete recovery.

The *cerebral complications* of the disease form a clinical picture that is uncommon. Recently Casparis has given a full description with case reports. The pyrexia, headache, vomiting, drowsiness, nuchal rigidity and Kernig's sign, one or all may appear and indicate an involvement of the central nervous system. Usually the symptoms supervene while the parotid swelling is at its height, although they may appear before the gland has enlarged or after the swelling has subsided. The spinal fluid shows a marked increase in its cell content. The lesion is probably a *meningoencephalitis* due to the presence of the mumps virus in the brain and meninges.

The *treatment* of mumps is guided by the fact that the disease is self-limited. Measures are to be directed largely to preventing the spread of the infection to other children in the family. Therefore, the patient is isolated for a period of at least 3 weeks in a well ventilated room heated at a suitable temperature.

It is customary to give a laxative, but this is no essential part of the treatment. If the fever is high, hydrotherapy is indicated for its control. Cleaning the mouth by sprays of mild alkaline solutions, such as a 1 per cent borax solution, is advised, or diluted alkaline antiseptic solution U. S. P. may be used. Proper cleansing of the mouth may prevent septic complications. The application of drugs to the glands is of no particular avail. Five to 10 per cent methyl salicylate in lard is recommended by some; this salve has a pleasant odor suggestive of healing. When there is pain and discomfort because of the tension of the swollen glands on their

fascial envelops, the application of an ice bag is often comforting. If cold is badly tolerated, warm applications may produce a soothing effect. For orchitis, elevation of the testes and scrotum and their support by a T-bandage is indicated. When the pain is extreme, light hot compresses are helpful; however, it may become necessary to give a hypodermic injection of codein ($\frac{1}{16}$ grain to a 25-pound baby).

The *treatment* of the cerebral complications is symptomatic; rest and lumbar puncture occupy a prominent place. When restlessness, insomnia and irritability are troublesome, a few doses of a mixture of 3 grains of sodium bromide (for a 25-pound child) in syrup of raspberry and water, equal parts to make a dram, given every 3 or 4 hours, may be helpful.

Smallpox

Smallpox is a disease little likely to be encountered during the infant period in this day of vaccination and careful public health supervision. The disease will not appear in a baby without a history of definite contact, unless it was derived from someone who had been infected with an exceedingly virulent strain of the disease and who had died before the appearance of the eruption, and had thus escaped diagnosis antemortem. One such case has come under our observation.

If smallpox has appeared in a family, immediate vaccination will protect all the contacts including infants. No delay should be permitted.

When the disease does appear, there is always a prodromal time of malaise and fever. During this preliminary period the baby will vomit and usually will have a diarrhea and show great prostration. In some instances, the child may die in this stage without developing any eruption. The prodromal fever is persistent and high without much remission until about the fourth day when the temperature drops to perhaps 99° or 100°. The fever does not rise above this point during the 3 or 4 days in which the rash is developing and passing from the macular through the papular and vesicular stages; but about the eighth day of the disease, when the vesicles become pustular, there is a secondary rise of temperature which reaches 104° to 105°. This fever persists until the dessication of the pustules is well advanced; defervescence goes step by step with the clearing of the skin.

The earliest evidence of the eruption is the appearance of a few

pinkish macules on the wrists. These rapidly become papules. The papules at first are hard and shotty; they are situated deep in the skin and are surrounded by an areola of swelling. The papules appear over the whole body about the same time, although a few can be found in and about the pharynx and palate before they are seen on the skin. Those that appear on the forearms, hands and feet may be delayed, but there is no cropping such as is seen in chicken-pox, nor is it possible in a given area to find various lesions in different stages of development. When a papular eruption appears, all lesions are papular; and when they become pustular, all are in that state in any given area of the skin. The uncovered parts of the body are more intensely affected in smallpox, and the covered areas in chicken-pox.

The chief complication that most concerns those who deal with children is septic infiltration with destruction of the skin, conjunctiva and cornea. The complications of the nervous system and heart are less frequent and less fatal in infants than in adults.

The *treatment,* apart from prophylaxis which includes isolation of the patient and vaccination of contacts, is directed toward supporting the child's strength by proper diet, the provision of fresh air and the comfort of good nursing. Tub baths are invaluable. In the San Francisco Isolation Hospital, iodine baths have been in use for many years and have proved to be of great value. Once a day, the patient is immersed in an iodine bath (½ to 1 dram iodine crystals to 5 gallons of water) and is left there for 10 or 15 minutes. This process limits the stage of pustulation and encourages healing. Following this treatment, septic complications, such as furunculosis, rarely occur, and as a result there is much less scarring.

The face is covered with a stockinet mask which carries a 20 per cent watery solution of ichthyol with ¼ of 1 per cent iodine. The results of this treatment of the face correspond to those following the use of the iodine bath on the body.

Scrupulous care must be exercised when pustules appear on the conjunctivæ. Frequent use of a 2 per cent borax solution for cleansing the eye, followed by the instillation of a freshly prepared 20 per cent solution of argyrol or a $\frac{1}{20}$ of 1 per cent zinc sulphate solution is indicated. The conjunctivæ should be irrigated with the borax solution at least every 2 hours, oftener if there is much secretion; and the antiseptic instillations should be used every 4 to 6 hours.

Ulcerations of the mouth and nasopharynx sometimes prove distressing. Alkaline sprays are to be used assiduously in order to keep the parts clean. Ulcerated points produced by the softening of the epidermis overlying the pustules are best dealt with by applying 15 per cent to 20 per cent silver nitrate solution on a cotton swab. Such an application should not be made too frequently; once daily for 3 or 4 days will usually suffice.

All bedding and personal linen must be put into a covered metal container partially filled with a 1 per cent or 2 per cent cresol solution and be boiled before they are washed. Food utensils and dishes, both of the patient and the attendant, should be washed in the sick room and sterilized by boiling before they are returned to the family kitchen. All excretions should be treated with chloride of lime or cresol before they are disposed of.

The patient should remain in quarantine for at least 3 weeks after desquamation has ceased.

Vaccination

Vaccination is a fundamental measure in the prophylaxis of smallpox. There is no choice for a medical adviser but to urge the necessity and insist upon the performance of vaccination for every child that comes under his care. A completely vaccinated community is a community positively protected against smallpox. Unfortunately, in this country we have been so well protected that we are becoming lax in our enforcement of vaccination, and children are allowed to reach the school age unvaccinated. Every child should be vaccinated not later than the end of its second year, preferably during the first year of its life. All the former objections to vaccination have been removed by modern methods of technic and preparation of virus.

There should be no reaction after inoculation until 3 or 4 days have passed. Then, at the site of the vaccination, a small red papule will appear; 24 hours later, this papule vesiculates and enlarges progressively through the next 4 or 5 days. During the stage of vesiculation, a reddened areola surrounds the lesion. From the seventh to the tenth day after inoculation, constitutional symptoms may supervene; these include anorexia, malaise, headache and fever. The temperature rise is usually moderate, but it may be extreme. The neighboring lymph glands are involved and at times become very tender. In the normal course of vaccination, the vesicle dries and a crust forms which loosens and becomes de-

tached after 10 days or 2 weeks. The area of a revaccination should be inspected at 24 and 48 hour intervals to determine the presence of an immunity reaction. Failing the appearance of this phenomenon, repetition of the inoculation should be insisted upon.

The appearance of a *generalized vaccinia* is characterized by the outbreak in different parts of the body of small vesicles, each of which becomes a pustule and follows a course similar to that of the vesicles of a mild variola. This phenomenon, however, is exceedingly rare after vaccination. Occasionally, a child may transfer the vaccine from the original inoculation, and a second vaccinal vesicle may develop simultaneously.

Indolent granulations that sometimes delay the healing of a vaccinal ulcer are best treated with 20 per cent nitrate of silver solution. These applications should be followed by the use of a piece of gutta percha tissue placed on the granulations and held there by the pressure of a firmly applied bandage.

Certain postvaccinal eruptions may be disconcerting. Of these, some may simulate scarlet fever or measles; others may appear as simple erythemata or as urticarias. Purpuric rashes are not unknown. Rarely tetanus has followed vaccination; in the great majority of these cases the vaccine is not the source of the infection, but the contamination comes from the finger nails of the patient.

An arm even slightly sore is exceedingly rare when the technic of vaccination is properly carried out. Tender, swollen arms frequently result from the use of the celluloid shield which is an apparatus worthy only of the utmost condemnation. No better culture chamber was ever devised than one of these shields closely applied and covered.

The site best adapted for inoculation is the outer surface of the arm. In the case of girl-babies, there is no reason why the extensor surface of the thigh should not be selected for vaccination although the posterior surface of the arm may be chosen for here the scar will be inconspicuous. After the inoculation, if a simple loose dressing of sterile gauze is applied and this is held in place by adhesive plaster strips, there will be no secondary infection and no pain due to mechanical pressure such as follows the use of the shield. In fastening the gauze dressing, it is important that the adhesive straps do not encircle the arm completely or in any way interfere with the circulation; if they do, venous engorgement and swelling with pain may occur, and

sloughing at the inoculation site may result. The primary dressing may remain on the arm until after the seventh day.

When there is no question of unclean linen on the part of the patient, dressings may be omitted. It is well, when this is done, to order that a change to fresh underwear be made daily. Impetigo may become implanted on a vaccinal lesion. It yields to treatment with 1 per cent ammoniated mercury ointment. Pyogenic complications, including erysipelas, are best treated for a few days by compresses saturated with equal parts of alcohol and glycerin. When the swelling and purulent discharge have abated, dusting powders of aristol or 10 per cent calomel in stearate of zinc may be applied.

Chicken-Pox

Varicella is a common disease among infants, but it is not often a severe one. It is usually easily differentiated from smallpox by the fact that the prodromal malaise and fever last never more than a day or two and that there is no period of intermission between the malaise and the appearance of the eruption.

The eruption is characteristic, and no name better describes it than the term, "glass pock," popularly used in Europe. Early in the vesicular stage, the lesions look much like small glass beads dropped upon the skin. Preceding this stage, macules and papules appear from which the vesicles develop. Later, the contents of the vesicles become turbid; soon the vesicles break and a blackish, central crust is formed. Lesions are often found on the scalp hidden by the hair, and others almost always appear on the hard or soft palate. Cropping is characteristic, so that in any given area of the body, when the disease is fully developed, the observer will find macule, papule, vesicle, pustule and crust—a state of affairs which readily differentiates the eruption from that of smallpox. Furthermore, the lesions seem to lie on the skin and not deep in that tissue as they do in the more severe malady. Chicken-pox is a self-limited disease with a course of from 5 to 8 days. The outbreak is usually limited to 2 or 3 crops of the lesions, but as many as 8 or 10 series of lesions may appear when the intensity of the infection is great.

Occasionally, there will be a prodromal rash which is remarkably like the rash of scarlet fever; this eruption may last from 1 to 3 days and disappear with the outcrop of the true varicella lesions. The cases in which this rash is encountered are

usually quite severe. The patient is ill, the fever is high and the true varicella lesions may be followed by extensive sloughs. Sometimes such patients develop septicemia and die. The phenomenon is probably due to a coincident infection with the streptococcus. It is in this type of case that acute hemorrhagic nephritis is most apt to occur, but nephritis may come on even in a mild case, either during the course of the eruption or afterward. Transient synovitis has been seen in the course of chickenpox, but it has no importance as a symptom. Hyperpyrexia of 1 or 2 days' duration is a rather common phenomenon of the disease during infancy, although most cases run an afebrile or slightly febrile course.

Treatment consists of isolation of the patient for the protection of other children, light diet, and if desired the use of a mild laxative, and of local applications to the lesions. Because of the excessive itching, the patient may scratch the lesions and produce an infected wound with consequent scarring. This symptom is best met by the use of a daily cresol bath, ½ to 1 dram to the gallon, and by the application to the pustules of a mixture containing coal tar solution (liquor carbonis detergens) dram 1, or phenol, dram ½ to 6 ounces of calamin lotion. This mixture should be shaken, poured out and applied with a cotton dab. When the lesions are definitely infected, touching each of them with 5 per cent iodin in glycerin and water helps to limit infection and prevent deep destruction of the skin and scarring.

Typhoid Fever

During infant life, typhoid fever may occur as a result of infection from contaminated food, especially milk; from a faulty water supply; from case contact; or from contact with a typhoid carrier. The disease is not particularly rare at this time of life. Griffith estimates that 1 patient in 25 who suffers from typhoid fever is under 2 years of age.

The long prodromal stage of adult typhoid fever is often absent in infant cases. Often the onset is with a high fever, which may be intermittent or remittent, together with vomiting and diarrhea which may be mistaken for evidences of an ordinary gastro-intestinal disturbance. The temperature curve in typhoid fever, as it occurs in infancy, runs a course different from that typical of the temperature line in adult life. The onset of the fever is sudden. At first, it may be quite high and then re-

mit, and afterwards, during the first week or 10 days, it rises steadily with a change to intermittence and the development of a wider diurnal excursion. A few patients, for the most part those in whom the disease is accompanied by profound cerebral symptoms (meningism, convulsions or coma), exhibit persistent hyperpyrexia for many days, sometimes even for 2 or 3 weeks. This temperature course is commonly seen in those cases which later prove fatal.

At the onset of the disease, convulsions may occur, but not so frequently as with some other febrile disorders. Meningism is probably more common in typhoid fever than in any other disease except lobar pneumonia and measles. In older infants, nocturnal delirium may be so marked that it becomes alarming. In some cases, the clinical picture is obscured by a persistent cough with or without a mild bronchitis. Rose spots, small pale red macules, or infrequently papules appear quite early in the disease; they are distributed over the chest, abdomen and back. Individual rose spots are very fleeting and disappear rapidly, to be replaced by others—a point of some diagnostic significance.

The spleen is rarely more than just palpable in the early stages of the disease; after the third week, however, it is usually quite large. The circulatory system does not respond as it does in later life. Slowing of the pulse and dicrotism are practically unknown at this age, while on the other hand the pulse is usually rapid and irregular. There is never that marked leucopenia which is characteristic of adult blood pictures. A count of 12,000 to 14,000 white blood cells does not negative the probability of typhoid fever in an infant. The Widal reaction is present in probably 90 per cent of the cases after the end of the first week. In those cases in which the agglutination reaction fails, the typhoid bacillus can usually be demonstrated in the stools. The appearance of the stools passed by an infant with typhoid fever is not characteristic. Hemorrhage following slough and perforation of the bowels is so infrequently reported as to appear a medical curiosity. Should it occur, the treatment is in no way different from the treatment of that complication as it appears in adults.

The course of typhoid fever in infancy varies little from the course run by the disease when it attacks adults except for the fact that the prodromal stage is so equivocal that it is frequently overlooked. The result is an apparent shortening of the disease.

At the age under discussion, generalized tuberculosis may sometimes present a clinical picture superficially similar to the

picture of typhoid fever. The blood findings are very much the same, but laboratory tests (Widal reaction and stool culture) ordinarily will clear the diagnosis. The onset of the two diseases is quite different. The long period of irritation, change of temperament and lethargy of generalized tuberculosis are absent in a typical case of typhoid fever. In those unusual cases with profound toxemia that sometimes occur in the course of enteric fever, irregularities of respiration and pulse are encountered, but they are never of the grouped type as they usually are when they form a part of the picture of generalized tuberculosis. The involvement of the cranial nerves, which occurs sooner or later in nearly every case of generalized tuberculosis in infancy, is hardly ever seen in a case of enteric fever.

The *management* of an infant patient with typhoid fever is essentially a matter of hygiene and diet, although drugs are useful adjuvants. Hydrotherapy is often of value in combating fever.

The importance of good nursing as an aid to the physician in the treatment of typhoid fever cannot be exaggerated. No physician is able to maintain intelligent control of a case without the careful daily study of a well kept record which shows the temperature curve and the various events of the disease in detail as they occur. As in any other severe disease, the sick room should be easily ventilated and well lighted. Strict cleanliness is the greatest of therapeutic agents. The patient's bedding should be changed frequently; this task is rendered easier for the nurse and more comfortable for the invalid if two beds are provided, so that the patient can be transferred from one to the other without much disturbance. When present, the teeth should be kept clean, and scrupulous care must be given to the toilet of the mouth. Care must be exercised not to damage the epithelium of the mouth by vigorous attempts to scrape the tongue and swab the buccal surfaces. A mild alkaline lotion sprayed from an atomizer into the mouth is the most effective and comforting way of cleansing that cavity. After every evacuation, the buttocks should be washed with a weak solution of green soap; following this, the nurse's gloved hands should receive a bath of antiseptic solution, preferably 1 per cent cresol.

The soiled bed-clothing and the personal clothing of the child should be dropped into a metal container, preferably a wash boiler, containing 2 per cent cresol solution. This container should be put on the stove and the contents thoroughly boiled before they are removed. All dishes used by the patient and nurse

should be sterilized by washing in hot water and soap, and they should be boiled before they are removed from the room, if that is possible.

Urine and feces should be treated by the addition of chloride of lime or antiformin to the vessel in which they are passed. Time should be allowed for the proper action of these chemicals before the evacuations are finally disposed of.

The management of typhoid fever has been made much more effective since the introduction of the high calorie diet. Under this method of feeding, many patients can be brought through the disease without loss of weight and with very little toxemia. The old idea that the intestinal canal of the typhoid fever patient is incapable of digesting and absorbing food and that the presence of anything but fluids in the tube increases the possibility of perforation has been thoroughly disproved. DuBois, working with typhoid fever patients fed by Coleman's method and checked by normal controls, found that in typhoid fever the protein loss is 10 per cent (normal 7 per cent to 9 per cent); the fat loss, 8.8 per cent to 15 per cent (normal 2 per cent); and the carbohydrate loss, 9 per cent (normal 0.1 per cent to 0.3 per cent). Clinical observers have found that the course of the disease is shortened by the use of the high calorie diet, that the chance of perforation and hemorrhage is decreased and that the patient comes out of the ordeal without the emaciation that follows older methods of treatment.

In the study of the fecal flora in typhoid fever, Torrey was able to demonstrate that some cases which ran a bad clinical course had a preponderance of proteolytic bacteria in the intestines. In such cases, the patient did badly on an exclusive milk diet, but when a high carbohydrate feeding was given, Torrey found that the toxic symptoms abated, and at the same time the concomitant bacterial flora changed from the proteolytic to the carbohydrate splitting types. He attributed the lessening incidence of intoxication to this change rather than to any alteration in the character or number of the invading typhoid bacilli.

It is estimated that healthy infants in the second half of the first year will need about 40 calories per pound per day in order to maintain them at weight. In the second year, 34 calories per pound per day will suffice. During a long-continued febrile period, another 10 calories per pound per day should be allowed in order to compensate for the increased waste of tissue due to the augmented metabolic processes. The diet should provide sufficient

protein to maintain the nitrogen need of the patient which will be higher than if the child were well.

Tympanites, a complication present in many cases, may be due to abnormal bacterial inhabitants of the intestine. Often these are spore-bearing anaerobes that thrive on protein but also readily split up carbohydrates to produce much gas. Dietetic measures useful in the control of tympanites of bacterial origin include a preliminary purgation with 2 to 3 drams of castor oil, followed by one day's restriction to a water or weak tea diet; and a second day's limitation of the intake to ½ or ¾ strength protein-milk, followed by a second dose of castor oil. Twelve hours later, a high calorie diet appropriate to the case is resumed. It must not be forgotten that often tympanites is not of intestinal origin but is an index of a failing circulation, a failure that may result from the continued toxemia or that may be the first sign of approaching dissolution. If the fever be controlled, often the tympanites will abate.

For purposes of *dietetic treatment,* it is good practice to consider whether the type of infection be mild, toxic (proteolytic), or fermentative (with diarrhea and acid stools).

For the first class, the **mild,** milk reinforced with sugars, dextrins and carbohydrates form the basis of the dietary. It is easy to estimate and order the amount of food that the child should have, by consulting a table of caloric values, remembering that a child of 2 years suffering with typhoid fever requires about 40 calories per pound per day to meet its energy needs, normal and abnormal. It will probably be necessary to begin with an intake much less than will supply the patient's energy needs and gradually to increase the feedings. The physician may be obliged to reduce the food from time to time in order to meet individual peculiarities. Of the available sugars, maltose, lactose and saccharose are most useful; of the dextrins, malted milk, corn syrup, dextrinized flour, flour ball, pulverized toast or zwieback crumbs may be employed. Of other carbohydrates, well cooked barley, wheat or rice flour will serve. Fruit jellies and fruit purées reinforced with milk sugar make excellent appetizers; given in moderation, they have little or no effect in producing diarrhea. Children relish an egg orangeade given once or twice daily (see Recipes).

In the **toxic** cases, milk is best withheld. For the first 24 hours, the only food allowed should be a weak cereal decoction, a high calorie orangeade (see Recipes), or a 10 per cent solution of lactose in water. Later, water-ices or iced fruit syrups reinforced

by lactose or zwieback, thin toast and cereal-jellies may be allowed. These foods should be given exclusively until the toxic symptoms abate. The child may then be put back upon the diet appropriate for cases of the mild type.

In the third class of case, the **fermentative**, with acid diarrhea, protein-milk (see Recipes) is quite the best form of food. Beside the protein-milk, egg white and meat decoctions may be given for the sake of variety, and after a few days boiled milk may be tried. To the diets of older infants who have passed the height of the disease, cottage cheese and scraped meat patties may be added cautiously. As the symptoms of fermentative diarrhea subside, the sugars and carbohydrates may be gradually added until the child resumes a diet similar to the one outlined for the mild type of case.

The following list of caloric values is useful in estimating the proper diet:

Whole milk, 1 ounce	20 C.
Cream, 20%, 1 ounce	60 C.
Protein-milk, pint	185 C.
Egg, whole	80 C.
Egg white	30 C.
Egg yolk	50 C.
Lactose, 1 ounce	120 C.
Saccharose, 1 ounce	120 C.
Dextrimaltose, 1 ounce	120 C.
Barley flour, 1 ounce	100 C.
Wheat flour, 1 ounce	100 C.
Rice flour, 1 ounce	100 C.
Butter, one pat	80 C.
Slice toast	80 C.
Toasted cracker, 1 ounce	114 C.
Cereal (cooked), heaping tbs.	50 C.
Apple sauce, 1 ounce	30 C.
Orange juice, 1 ounce	15 C.

For the control of the fever, the tympanites and the nervous symptoms, there is no procedure comparable to the application of the tepid pack. A sheet wrung out of water at 95° to 100° is wrapped around the child, care being taken that it is tucked in well around the arms and between the thighs. A blanket is then wrapped over the sheet, and the infant is left in the pack for from 10 to 15 minutes. The blanket may be raised now and then and a little cold water sprayed onto the sheet. It is sometimes necessary to pack the child 4 or 5 times in 24 hours, especially if the temperature is persistently high and if the nervous symptoms are distressing. No harm can follow the continuous use of the tepid pack for from 8 to 10 hours and sometimes it may be of

advantage to leave the child in the pack for as long as 24 hours. The patient may be changed from one pack to another every 2 or 3 hours. Such a drastic procedure will rarely be necessary and then only when hyperpyrexia of itself, is alarming. Packs are well borne and disturb a child very little. For this reason they are preferable to tub baths. Sponging with tepid water is permissible for cases in which the fever is slight or for cleansing purposes. In order to be efficient as an antipyretic measure, it must be continued for a considerable period of time, and infants usually protest vigorously against its use and become restless and irritable. A detailed description of the preparation and use of the pack is given in the chapter on Methods.

If the bath is employed, continuous friction to the patient's skin must be used even when the temperature of the water is as high as 100°. Under these circumstances, cool towels should be applied to the head while the child is in the water. If hydrotherapeutic measures are followed by definite circulatory depression with a weak pulse and cyanosis, it is best not to return to their use. Except in extraordinary instances, the fever alone will do the child little harm and it will disappear without treatment as the toxic symptoms abate in the normal course of the disease.

There are a few *drugs* that find a legitimate place in the treatment of this disease. For the most part, they are the cardiac stimulants. Experienced clinicians agree that moderate doses of alcohol in some form are of benefit. The simple elixir of the pharmacopeia is an efficient and obtainable alcoholic stimulant. It is of greatest value when nervous exhaustion with a weak pulse and blurring of the first heart sound occur. Given early, in the absence of these warning signs, its proper use may prevent their appearance. For a 15-pound baby, 10 to 15 drops of the simple elixir diluted in a dram of water may be given every 3 or 4 hours; older and heavier children may have proportionate doses. If either brandy or whisky is used it should be given in 8- to 15-drop doses diluted in water for a 15-pound baby. Alcohol is a drug and it should not be added to the child's food.

When cardiac insufficiency threatens, tincture of digitalis is valuable in doses of from 2 to 3 minims. In an emergency, the intravenous injection of strophanthin ($\frac{1}{1200}$ to $\frac{1}{800}$ of a grain for a 15-pound child) is of great value because of its prompt action on the heart; this can be followed by the use of tincture of digitalis. Caffein-sodium-benzoate is a combination usually found in the physician's hypodermic case and is an efficient drug with

no bad after-effect; 1/8 of a grain by hypodermic or 1/4 of a grain by mouth may be administered every 3 or 4 hours. When exhaustion is great and collapse threatens and the failure of the circulation is impending, there is no drug that approaches camphor in promptness and efficiency of action. It is advantageous to use larger doses than are usually given. Because of the fleeting effect of the drug, early repetition of the dose is indicated; 1/4 to 1/2 grain of camphor in olive oil or in ether can be given at 1/2 hour intervals until as much as 2 grains have been given to a 15-pound baby. Strychnin is a drug that should have no place in this or in any other disease in which there is a tendency to nervous irritability.

Hemorrhage from the bowel is so rare an accident to an infant with typhoid fever that it need only be mentioned; in general treatments applicable to the adult patient apply here. An opium derivative may be given in a dose appropriate to the weight of the child; it ought to be large enough to keep the patient drowsy for some hours and should be repeated often enough to maintain the condition through 1 or 2 days.

The signs of perforation are to be watched for with anxious scrutiny and if they appear, as happily they do rarely in infant patients, immediate surgical intervention is indicated.

Constipation is not often troublesome in the typhoid fever of infants. It is better to treat it by carefully given enemas than by purgative or laxative drugs, although no harm can follow the daily ingestion of 1 to 2 teaspoonfuls of a heavy petroleum oil.

Diarrhea, when it occurs, is usually slight, and when moderate, no attempt should be made to treat it. The more severe types of diarrhea with watery stools often respond to dietetic treatment, particularly to the withdrawal of milk. If after a few days this measure fails to check the excessive evacuations, small doses of opium may be used for a short time. The deodorized tincture, 1/4 to 1/2 minim in water, may be given a 15-pound child for 1 or 2 days at 4 to 6 hour intervals. It is important that a child never should be wakened for opiate medication. Should a tympanites occur while the opium is being given, the drug should be withdrawn, as there is reason to believe that occasionally it produces that condition.

Vomiting is a rather frequent complication of the early stages of enteric fever; it is rarely intractable and it responds promptly to dietetic measures. The withdrawal of milk and the use of high

calorie orangeade, or of weak broths and cereal decoctions are valuable weapons against this symptom. The influence of drugs in producing vomiting must not be forgotten and the fewer drugs used in the course of typhoid fever, the better.

The pulmonary complications are rarely such as to demand treatment other than a well ventilated room and the occasional inhalation of steam. These complications are essentially a part of the pathologic manifestations of the disease and are self-limited, disappearing gradually as the child acquires immunity. Much harm may be done by the overtreatment of the pulmonary complications of typhoid fever. Even when a lobar pneumonia appears as a part of the clinical picture, expectant treatment is more fruitful than pharmaceutical procedure.

During the convalescent stage, management is important; undue haste in getting the weakened child out of bed must be avoided. Because of the exhausting nature of the disease, the environment must be kept as tranquil as possible and no undue mental or physical strain allowed. On the other hand, when the child is in fairly good condition, it often happens that a slight fever persists and remains for some time after all the other symptoms have disappeared. Under these circumstances, the patients are usually benefited if the presence of the fever is ignored and they are taken from their beds and allowed to be in the open air for a time every day.

Often the effect of the long-continued toxemia leaves the heart irritable and the pulse rapid and irregular. These signs of cardiac instability may run on for weeks or even months after the cessation of other symptoms. The heart itself is rarely at fault and if the child is permitted to leave its bed under careful supervision, it will be found that fresh air and sunshine are the most effective therapeutic agents.

Pertussis (Whooping Cough)

Pertussis is a very serious malady in infancy. When it attacks babies in their first year, it is alarming and often fatal. The disease begins without any characteristic expression, and a period of 10 days or 2 weeks elapses before the pathognomonic whoop appears. This symptom may be absent in the case of young babies or in older infants who are weakly or who have acquired a complicating bronchitis or bronchopneumonia. During the stage of onset, the child at first exhibits a mild coryza and

a slight cough, staccato in character. This cough is infrequent during the day but from the beginning is more frequent and more severe at night. After 4 or 5 days, although the whoop does not develop, the staccato effect of each paroxysm is increased and a certain amount of asphyxia accompanies the cough. This is made evident by the general discomfort of the child, reddening of the face and watering of the eyes. About the tenth day, at the end of such a paroxysm, the unmistakable whoop will be heard, except in the case of very young or weakly babies. The whoop is produced by a long-drawn inspiration through a narrowed glottis; it follows a series of ineffective coughs, during the progress of which the child's face becomes suffused. In a young infant an alarming degree of cyanosis and asphyxia may supervene. It is when such recurrent attacks of asphyxia develop without any whoop that the prognosis is worst.

Usually a day or two before, but often not until a few days after the appearance of the whoop, the paroxysmal attack will be followed by vomiting. In young and in poorly nourished infants, the persistence of emesis following paroxysmal cough with asphyxia is diagnostic of pertussis even if no whoop is heard.

In the paroxysmal stage of the disease, from 5 to 50 paroxysms may appear during 24 hours. The duration of this stage varies from 10 days to 2 or 3 weeks. The attacks become progressively less in number and milder in character until they cease to be constant phenomena, and the stage of defervescence is entered. During this period of the disease, bronchial irritative symptoms are fairly constant. The typical paroxysmal attacks occur infrequently and then only when they are induced by some emotional stress.

If the paroxysmal cough persists for more than 2 or 3 months after the beginning of the paroxysmal stage of pertussis, it is certain that there is some cause for the persistence other than the original infection; most often, an involvement of the mediastinal or bronchial lymph glands is responsible. The enlargement of these glands may follow simply from the pulmonary effects of pertussis, or it may be evidence of a tuberculous invasion that has been favored by the influence of the pertussis on the pulmonary lymphatic tissues. In older infants, it is possible that the habit of paroxysmal cough may be established, and that the cough in reality is but a form of tic.

At any time after the onset of the disease, hemorrhages may

appear; these are usually slight and most often are limited to the conjunctivæ and to the nasal mucous membrane. Infrequently, petechiæ are seen. Very rarely, an intracranial hemorrhage, either cerebral or meningeal, takes place. After such a hemorrhage, a spastic paralysis, usually a hemiplegia, is almost certain to develop. These are unusual complications.

The ordinary complications of pertussis are largely referable to the lung. A slight degree of emphysema is present in every case after the paroxysms begin. At some time during the course of the disease, evidences of bronchitis are present. Usually this bronchitis is confined to the large tubes and is of little significance; but when, as sometimes happens, the small tubes and the air cells become involved in the development of a bronchopneumonia, the condition is one that must cause anxiety.

Aside from respiratory involvement, affections of the gastrointestinal tract are the most harassing accompaniments of pertussis. The vomiting is less a complication than it is a symptom but the nutritional disturbances which follow it are of great importance to the child, especially if it be a young infant. Methods for the maintenance of nutrition must be carefully considered and persistently applied. Diarrheas, both infective and nutritional are apt to accompany the more severe cases of whooping cough, and their prevention and treatment often provide perplexing therapeutic problems.

There is no natural immunity to pertussis. The only way to prevent its development is to keep the child out of contact with other children who are affected. Apparently the disease is highly infectious during the stage of onset, before the typical symptoms appear, than it is at any other time during its course. Therefore, it is at this stage that a pertussis patient is most dangerous to others.

An infant may become infected by another child or by an adult who brings the disease into the house. These two facts, taken together with the knowledge of the particular danger of the disease for babies, make it essential that every infant be well isolated and protected from contact with school children and other visitors, especially those who cough. If mothers and nurses could be taught the dangers of hand infection, infants could be protected, not only from pertussis, but also from many other respiratory infections. No one should be allowed to fondle or kiss babies' hands, and it would be well if the attendants could habituate

themselves not to touch the child without first washing their own hands.

There seems to be little doubt that the bacillus isolated by Bordet and Gengou is the true infecting agent of pertussis. This bacterium is found abundantly in the mucus secreted by the respiratory tract during the stage of onset and, less certainly, later. The blood serum of patients who have recovered is effective in agglutinating the organisms. These facts give a logical reason for attempts at immunization and cure by the use of a specific vaccine prepared from this bacillus.

If a diagnosis can be made before the onset of the paroxysmal stage, much can be done to minimize the rigors of that period. It is possible that the use of a reliable, freshly prepared vaccine may cut short the trouble before the onset of the paroxysms; probably it will minimize the frequency and severity of the attacks. Curiously enough, the vaccine is more effective for young infants than it is for older children. For a 15-pound infant, ½ c.c. of a vaccine containing to the c.c. 250,000,000 of the Bordet-Gengou bacillus, 125,000,000 each of staphylococcus albus and aureus, 100,000,000 each of B. influenza and pneumococcus and 200,000,000 of micrococcus catarrhalis is sometimes effective. This dose is to be repeated every second day until 4 injections are given. It is probable that vaccines do little good after the paroxysms are well established; but if they are given to a child that has been exposed to the infection and who is just beginning to cough, the paroxysmal stage may be aborted or at least its severity may be materially reduced.

When the staccato character of the cough, its tendency to become paroxysmal, its greater frequency at night and the appearance of emesis, lead us to suspect the stage of onset of whooping cough, the use of antispasmodic drugs as well as of vaccines is indicated. No remedy has stood the test of time better than antipyrin. However, because of its depressant action, this drug should be prescribed together with caffein sodium benzoate. The addition of bromide of soda and spirits of chloroform reinforces the antispasmodic action of antipyrin. A mixture somewhat as follows may be made up: antipyrin, grains 8; caffein sodium benzoate, grains 4; sodium bromide, grains 20; spirits of chloroform, minims 2; glycerin, drams ½; and chloroform water, to make 1 ounce. Of this 30 drops in water may be given to a 15-pound infant every 3 hours.

When the night's rest is interfered with, heroin or codein with atropin are of value. They should be given in small doses, $\frac{1}{100}$ grain of heroin, or $\frac{1}{40}$ grain of codein and $\frac{1}{800}$ grain of atropin, repeated once, if necessary, after 4 or 6 hours. The opiates should be used only under the stress of necessity and then but for a few successive nights. It is much better not to give these drugs during the day.

There is no doubt that depressions of temperature, cold drinks, and draughts of cold air blowing across the face of a pertussis patient, will increase the frequency and intensity of the paroxysms. Therefore, it is well to have the sleeping room kept warm and the air moist, and while not neglectful of the ventilation, to arrange screens so that direct draughts of air may not beat on the patient. It is a well authenticated observation that children who spend their days in the open air have less distress during the course of pertussis than those who are kept indoors. It is better, when possible, to send the patient to a warm climate, free from winds, dust and fogs, in which he can be kept constantly outside.

During the paroxysmal stage, vomiting is a distressing and sometimes an alarming symptom. Children may reject all food and lose in weight. In a few cases starvation becomes so extreme that the acidosis of food deprivation ensues. Semisolid, gelatinous foods are better retained by vomiting individuals than either liquids or solids. This fact can be utilized in arranging the dietary for children suffering from pertussis accompanied by vomiting. It will be found that cereal jellies, custards, junkets, broths thickened with sago or flour, and gelatin puddings are well retained. For young babies, thick formulas, such as are used in pyloric stenosis and habitual vomiting, will be of service (see Recipes). A feeding given shortly after a paroxysm with emesis will often be perfectly retained. For this reason, it may be necessary to abandon any attempt at regularity in feeding and give the meals whenever the child is able to retain them. In combating vomiting, we must depend entirely on dietetic measures, for drugs are of no avail in checking the tendency to emesis.

Certain infants, most of whom suffer from spasmophilic tendencies, may develop severe spasm of the larynx during a pertussal attack. They may lose consciousness and go into asphyxial convulsions. At the height of the disease, such a child may have from 10 to 100 of these seizures during a single day. This com-

plication, which is very alarming to see, best can be met by the use of a tongue forceps such as an anesthetist employs. At the onset of a seizure, the tongue should be drawn out and rhythmic tractions made at the rate of 16 to 20 per minute. If this is done promptly, the full development of the seizure may be prevented.

We have used on occasion the therapeutic measure recommended by the French—the intramuscular injection of ether in doses of ½ to 1 c.c. In certain cases it seems to be of value, particularly if there is a complication of bronchopneumonia.

Recently the quartz lamp therapy has come into vogue. In those cases which have come under our observation, the most outstanding result has been the marked improvement in the children's general condition which enables them better to deal with the infection.

In the stage of defervescence, the hygienic measures are still necessary. The time for the use of antispasmodics has passed, but quinine becomes a remedy that may do good. One of the tasteless forms of the drug (euquinine) may be used 4 times a day in ¼ grain doses for a 15-pound child. Chocolate syrup makes a pleasant vehicle for its administration.

The postpertussal paroxysmal cough that remains with so many infants is rarely annoying. It probably results from some enlargement of mediastinal glands and usually ceases after a few weeks or months. No therapeutic means available to us is very effective in checking it. The iodids sometimes prove of value. Syrup of ferrous iodid, minims 5; glycerin, minims 5; and chloroform water enough to make a dram, every 4 hours, is a reputed prescription. When the cough is harassing enough to interfere with sleep and nutrition, the occasional use of codein, $\frac{1}{40}$ grain, or heroin, $\frac{1}{60}$ grain, with atropin, $\frac{1}{600}$ grain, for a 25-pound child may become necessary. Fortunately this postpertussal cough is self-limited. If it persists over 5 or 6 months after the end of the paroxysmal stage, we must suspect the presence of mediastinal gland tuberculosis, a suspicion which should be tested at once by the scrutiny of an x-ray picture of the patient's chest.

The *treatment of lung complications* of pertussis differs in no way from the treatment of pulmonary affections as they occur apart from the disease. In the bronchopneumonias, the mustard pack is invaluable; other forms of hydrotherapy are useful, especially when hyperpyrexia is a feature. Of drugs, atropin is useful,

particularly when there is a spasmodic element in the bronchitis or a bronchopneumonia; it also has definite value in lessening the intensity of the pertussal paroxysm.

When the pulmonary complications lead to circulatory depression, camphor (10 per cent in olive oil) is a remedy that can be depended upon. It should be used in doses, 8 to 12 minims, given by hypodermic injection every 2 hours. The treatment of a lobar pneumonia with pertussis is essentially expectant with careful attention to maintenance of cardiac competence. For the emphysema there is no treatment, neither is there any way of influencing the bronchial gland enlargement that sometimes follows the disease except by the provision of the best hygienic surroundings, the use of a generous dietetic regime and when possible recourse to heliotherapy. (See Methods.)

In order to prevent the spread of the disease, it is essential that the patient should be kept from contact with other children for a period of 3 to 6 weeks after the paroxysmal cough has ceased. It is not necessary to confine the child to the house during this period. Whenever it is possible the child should be out of doors during the day, whatever the stage of the disease. Nothing except the most severe complications should prevent this measure.

Influenza

Contrary to the general opinion, infants are frequently attacked during an epidemic of the disease commonly known as **influenza**. At the Children's Hospital in San Francisco during the 1918-1919 epidemic, nearly 800 children of all ages were admitted with this disease, of whom 20 per cent were under 2 years of age. Apart from cases occurring in an epidemic, the disease is rarely found in infants.

Infants may respond to the infection by a great variety of symptoms with the result that the clinical picture may vary from a mild malaise to the most severe seizures with pulmonary involvement, progressive gastrointestinal disturbances or profound affection of the nervous system.

Ordinarily, the symptoms are respiratory. The disease may affect any or all parts of the upper or lower respiratory tract, with the result that the respiratory symptoms are dominant. Coryza, pharyngitis, laryngitis, bronchitis, bronchopneumonia, or true lobar consolidation, with dry pleurisy, may appear. A

purulent, pleural effusion occurs as a sequela in 1 or 2 per cent of the patients who show pulmonary involvement. All of these symptoms may occur simultaneously or in sequence, or any of them may happen in combination—a result that makes the diagnosis of the respiratory conditions of the disease perplexing.

Much of the restlessness and irritability which accompany this disorder is due to inefficient circulation. Many of the infant patients, especially those who have acquired massive lung complications or profound toxemias, show dilated right hearts and true irregularity of the pulse with feebleness and irritability. In such instances, cyanosis is marked, although in a few extraordinary cases of cardiac failure, pallor will be predominant.

Either alone or accompanying the respiratory symptoms, vomiting may occur; it may be either mild, or persistent and intractable if the infection is one of great severity.

Infants with influenza are always restless and irritable. In some mild cases and in many of the more severe, meningism with head retraction and nuchal rigidity is present. Convulsions may usher in the abrupt onset of the disease. In older infants, and in certain of the younger, lethargy and stupor akin to coma may continue for several days. *Encephalitis* may follow an attack of influenza or it may sometimes entirely replace a usual attack of the disease. Infants may fall victims to that peculiar form of basal encephalitis which has received so much attention within recent times and which has been called "encephalitis lethargica." Meningitis, with the presence of Pfeiffer's bacillus in the spinal fluid is a rare but well recognized sequela of the infection. There are on record cases of myelitis which have resulted in spastic paraplegia. Multiple neuritis, while very uncommon, has also been observed. These complications receive fuller discussion elsewhere.

The course of the fever is characteristic. Usually the curve is an irregular one ranging from 104° to 106° from the beginning of the disease. The younger the patient, the higher the temperature elevation is apt to be. In older infants, the fever may be protracted over a period of several weeks. If this happens coincidently with marked gastrointestinal symptoms and without predominant respiratory involvement, it may be difficult to differentiate such an atypical case from typhoid fever; the final diagnosis will be greatly facilitated by proper laboratory procedure.

Even during infancy when the leucocytes are normally plenti-

ful, the blood picture of influenza is that of a leucopenia. In an uncomplicated case, 6,000 to 7,000 leucocytes per c.mm. is about the average, and sometimes they are found to run as low as 2,000. However, in those cases in which the pneumonias, especially lobar pneumonia, are features, high counts up to 25,000 to 30,000 with 60 per cent to 80 per cent of polymorphonuclear leucocytes are often observed.

The *streptococcus* is a frequent accompaniment of the specific virus of influenza. The result is that in many cases erythemas resembling the exanthemas, especially scarlet fever, may confuse the diagnosis for a short time. A few of these rashes closely simulate measles, so that great care sometimes is needed to make an accurate differentiation. Nephritis usually does not affect infants as a complication of this disease, but in the records of the San Francisco Children's Hospital there are notes of 2 cases of hemorrhagic nephritis which occurred in influenza patients under 2 years of age. However, some irritation of the kidneys with inflammation, evidenced by albumin in the urine, usually can be demonstrated. Certain upper respiratory infections, usually of streptococcic etiology, may give a picture remotely simulating an influenza, but these infections are ordinarily self-limited and of little gravity.

A large proportion even of the mildest cases of influenza, develop an otitis media; in a very small number of these, the mastoid cells are involved. The otitis is remarkable for its insidious onset and for its painlessness. Routine scrutiny of the ears at one examination may show perfectly normal ear conditions, and at another a few hours later, a bulging or ruptured membrane may be discerned, a state of affairs strikingly different from that found when otitis is a complication of any other infection. Cultures of the otitic pus invariably reveal a preponderance of pneumococci, together with a few streptococci.

Hemorrhages are common. Persistent epistaxis is a frequent complication; usually it is readily controlled by the use of pressure applied through cotton plugs. Bleeding from the stomach may be responsible for death in older infants, and diarrhea with bloody stools frequently occurs. Hematuria is rarely a part of a true nephritis. The characteristic hemorrhagic sputum of older patients fails to appear because infants are unable to eject sputa except when they vomit. Hemorrhages into the skin in

the form of petechiæ or as more massive lesions have been recorded.

The *treatment* in the mild cases is expectant. The fever, headache and nervous symptoms are best controlled by hydrotherapeutic measures, such as packs and sponging. When vomiting is present, washing out the stomach with a 2 per cent sodium bicarbonate solution is followed by prompt relief. It is rarely necessary to repeat this procedure more than once or twice. Many of these vomiting patients suffer as much from dehydration as they do from the toxemia of the disease, and it is well after lavage to leave some of the alkaline solution in the stomach. When the toxemia is extreme, intraperitoneal injections of from 150 to 250 c.c. of normal salt solution are of the greatest value. Proctoclysis may be used as a substitute, but the technic is difficult in the case of the very young patient. Hypodermoclysis may be resorted to, but it is less promptly effective and gives more shock than the injection of fluid into the peritoneum when this procedure is properly carried out. (See Methods.)

The diarrhea is met best by dietetic measures. An initial dose of castor oil (3 drams to a 25-pound child), or calomel (1 grain for a patient of the same weight), may be given. This should not be repeated; when there is blood in the evacuations, it is best to omit the purges. As the toxemia retrogresses, the diarrhea will abate. Many instances of diarrhea are due to concomitant abnormal proteolytic or saccharolytic flora in the intestine and the increased bowel movements will respond in the first case to a high carbohydrate diet and in the second to an augmented protein diet. Should constipation be a feature of influenzal infection, which it seldom is, it should be met by carefully given, low enemas of 5 per cent sodium bicarbonate solution. (See Methods.)

The normal diet for a child suffering from influenza will be at first fluid—high calorie orange juice, or milk augmented with lactose or cream-water-lactose mixtures. Later, for older infants, there may be added ice cream, water ices, egg-orangeade, strained cereals, fruit juices and fruit pulps and light broths carrying fine, well cooked cereals such as barley or sago. (See Recipes.) As the disease progresses favorably, the diet should be enriched by the addition of more milk, eggs and carbohydrate foods.

When the patient is lethargic or somnolent, the mustard pack is thoroughly effective in stimulating the sensorium and rousing

him. Lethargy is an evidence of an extraordinary toxemia and is met best by the free use of fluid by mouth or by enteroclysis, and in extreme cases by massive intraperitoneal injections of normal salt solution repeated at 6 or 8 hour intervals. It is often astonishing how promptly the lethargy and somnolence will disappear when water is given in adequate amounts. The use of 5 per cent lactose solution ingested by mouth and of glucose in equal strength solution administered by rectum have a nutritive value and possibly an antitoxic influence.

The ear should be kept under routine observation from the onset of the disease and at the earliest sign of tympanic involvement, a paracentesis tympani followed by proper cleansing of the ear is indicated. When it is possible, it is well to have these complications kept under the observation of a skilled otologist. This is advisable because of the insidious way in which mastoid involvement may develop.

Cough is a persistent and a distressing symptom which is usually more in evidence in the mild cases than in the severe; in these cases, most often it arises from local irritation in the pharynx. The typical brassy, choking cough of laryngitis is also heard when the larynx is involved, a condition of affairs that takes place more frequently during infancy than at a later age. With an uncomplicated pneumonia, cough is rarely extreme. The pharyngeal type of cough responds better to local applications than to cough mixtures. One or two applications of a 10 per cent solution of silver nitrate made promptly at the onset, does more to check the cough than anything else. With a laryngeal cough, inhalations of steam and the intramuscular injection of atropin or the use of occasional doses of codein with spirits of chloroform are indicated in the milder cases. In the more severe types, apomorphin or ipecac to the point of emesis may be necessary to clear the air passages of the tenacious mucus. Ice collars to the throat are of great value. The obstruction may be such that it becomes necessary to resort to intubation. Unfortunately, however, the swelling and inflammation is not limited to the larynx alone and the intubation tube may become plugged with secretions from the trachea. Even tracheotomy may be futile because of the membrane-like secretions that form below the lower opening of the tracheotomy tube.

The cough arising from pulmonary involvement, usually yields to hydrotherapeutic measures, such as the mustard pack or the wet pack. (See Methods.)

In certain of the complicating pneumonias, the cough is the result of cardiac weakness. There is dilatation of the right heart with a consequent passive pulmonary congestion added to the inflammatory events that have occurred in the lung. Children so affected are cyanotic, and nothing quiets their cough quite so well as an initial dose of strophanthin, 1/2000 of a grain for a 15-pound infant, given intravenously and followed by the proper use of digitalis. Opium is valuable; it is best given in the form of codein, or Dover's powder, or in the old Brown's mixture. It is especially useful when the cough is so harassing that it keeps the child wakeful and restless.

Dyspnea as a symptom recedes promptly with the subsidence of the toxic effects of the invasion, except in those cases where it is an accompaniment of cough or cardiac failure. The methods outlined for the easement of cough will be effective in many cases, and the proper support of the circulation by the use of strophanthus, digitalis or caffein are indicated in order to relieve cardiac types of dyspnea.

The treatment of bronchopneumonia as a complication of influenza calls for the same measures as are used in the ordinary bronchopneumonias—mustard packs, the iodides, stimulants and steam inhalations.

Many recommendations have been made for the control of the hemorrhages in influenza. Injection of serum, of healthy human blood, of coagulin and of cephalin have been tried without more success than might have been expected from a consideration of the type of blood dyscrasia that must occur in the presence of such an infection. In those cases which are not fatal at an early hour, the hemorrhages are self-limited and tend to disappear as the more toxic phases of the disease subside. Of drugs, the only one that is of any value is opium; its worth depends upon its power to allay restlessness and to quiet the circulation.

The prevention of the spread of the disease is a matter of difficulty because of the fact that for several days before the disabling symptoms appear, the infected individual carries the virus. The use of the gauze mask has been both applauded and condemned. There can be no question whatever in the minds of those who have watched a properly conducted experiment, that the mask worn with understanding and care, is distinctly a protective device. Certainly, nurses and physicians and other attendants on influenza patients should wear a mask while in contact. It must be remem-

bered that hand infection is exceedingly common and that scrupulous cleanliness of the hands should be insisted upon. The value of the mask as a community measure during an epidemic is open to some question, not because a properly devised mask, correctly worn, is not protective, but because it is impossible to enforce the use of an effective mask, effectively worn. Certainly for those who frequent crowds in street cars or other places, the mask is to be advised.

The same precautions that are used in typhoid fever cases should be urged on those who attend influenza patients.

Malaria

Malaria is more common in some parts of the country than it should be when we consider the efficiency of modern methods of mosquito control. When the effective suppression of the disease in the African colonies of France and England is remembered, there seems to be no reason why there should be malarial areas in the rich and comparatively closely populated and well educated United States.

The essence of the prophylaxis of the disease lies in the measures directed to mosquito destruction, a question in which every physician should be interested. The child itself should be protected from the insects by house screens and by effective mosquito netting about the crib.

When a microscope is available, the diagnosis of malaria should not be accepted as final until the plasmodium has been visibly demonstrated. In some parts of the country, many children who have septic infections with fever, with or without chill, are dosed with quinine to relieve a supposed malaria. This is especially the case with girl infants who suffer from pyelocystitis, a very common condition which is too frequently overlooked. Septicemia of streptococcic origin and subacute tonsillar infections are also often mistakenly treated for malaria.

Bass and Du Buys have pointed out the need that exists to search out and treat malaria carriers—individuals who may be heavily infected yet present no symptoms. Such persons may be sources of mosquito infection and indirectly be responsible for the spread of the disease.

The chill of malaria as it occurs in an infant is a very striking and somewhat alarming manifestation—quite different in appearance from that seen when an adult is the victim. The onset is

abrupt to an astonishing degree. The child who is playing, perfectly happy and apparently in the best of health, suddenly becomes deeply cyanotic, dyspneic and goes into collapse without loss of consciousness. The little one will lie down wherever it happens to be and remain motionless with closed eyes and an expression of anxiety. Occasionally the bowel and the bladder will empty themselves spontaneously. This combination of cyanosis, dyspnea and depression lasts from 5 to 20 minutes before the child recovers color and breathes more easily; but the infant remains obviously very ill until the stage of malaise passes off after a few hours. Except for increased pallor and anorexia, the child does not seem particularly ill in the interval between these rigors. Blood drawn at the time of the rigor will reveal many of the typical plasmodia.

During infancy, such characteristic manifestations may fail; instead the baby will exhibit diarrhea, vomiting or especially convulsions. These gastrointestinal symptoms together with apathy, increasing perhaps to coma, may simulate typhoid fever—a condition of affairs most frequent in estivo-autumnal infections. On the other hand, the fever of pyelocystitis (usual in girl babies) or localized empyema from other low grade sepsis may be confusing. In either case laboratory methods will clear the diagnosis.

If the physician is fortunate enough to observe the infant shortly before or during a rigor, this will be the time for the initial dose of quinine bisulphate or dihydrochloride in a syrup vehicle, preferably yerba santa. One-third to 2 grains, according to the age and weight of the child, should be given by mouth three times a day for four days, and thereafter, daily, for eight weeks.

When the child has a chronic form of the disease, a mutual immunity will have been acquired between the plasmodium and the infant. Under these circumstances, the chills are less dramatic in appearance than they are in the acute form of the disease; the malaise is more profound and there is a greater degree of anemia, while the spleen is much enlarged. In these chronic cases the plasmodium is often elusive.

Many malarial children have been treated with insufficient doses of quinine, as a result of which the plasmodium has acquired a toleration for the drug. The therapy of such cases has lacked either in duration or sufficiency of dose. The standard treatment outlined above has been recommended by C. C. Bass

and other authorities as entirely adequate, not only to cure, but to disinfect 90 per cent of patients infected with malaria. Bass discredits intramuscular therapy as painful and as less effective than oral. However, when quinine disturbs digestion, the dihydrochloride can be injected into the triceps muscle. The writers have found its use in this way effective and well tolerated by infants who show little distress after such injections.

For infants who are intolerant of quinine by mouth, British authorities on tropical medicine advocate the use of the drug by rectal injection. For its administration, the dose is an amount of the bisulphate or dihydrochloride of quinine twice as great as would be appropriate for oral administration to the patient. The drug is dissolved in an ounce of warm water; with the child's hips elevated, this is slowly injected through a short catheter into the rectum.

When possible, the convalescent child should be removed from the malarial district in which it lives. It must not be forgotten that every infected individual is a carrier of the disease and for the protection of other children in the community, the patient should be protected from attack by anopheles mosquitoes.

Erysipelas

Erysipelas occurs with moderate frequency during the period of infant life. It may be encountered immediately after birth or at any time within the first 2 years of life. The disease has the reputation of being deadly, especially when it attacks the newborn. Some authorities state that children attacked during the first 6 weeks never recover, but this has not been borne out by the writers' experience.

There seems to be definite variability in the intensity of the infection. Some patients, even among the newborn, respond promptly to treatment; others die rapidly with toxemia; while those of another group seem to acquire a partial immunity, sufficient to hold the symptoms in check but insufficient to terminate the disease. The persistent cases are the most difficult with which to deal. Sometimes when the child seems to have recovered, a new area of inflammation will appear suddenly and spread over the body. Periods of exacerbation and remission of this sort may last for several months before the disease ends in the child's recovery or more frequently in its death from exhaustion.

The initial lesion in erysipelas is always a wound. In the new-

born, it is most apt to be about the umbilical stump or in the regions of the vulva, in girls, or of the lower abdomen in boys. Occasionally a streptococcic conjunctivitis may usher in the disease. Often pin punctures and excoriations from one cause or another may provide a portal of entry for the streptococcus. More commonly than is generally thought, impetigo of the very young runs into the streptococcic lymphangitis which we call erysipelas.

The first sign of the disease is a red papule at the site of the inoculation; or in the impetiginous cases, a red areola spreading from a pustule or crust out into the surrounding skin. Redness and swelling advance rapidly with the typical raised, red edge. They spread circumferentially. In children the central part of the erysipelatous area blanches after a short time but remains swollen. With some infants whose skins are exceedingly fine, it is difficult to differentiate an erysipelatous blush from a simple erythema by inspection alone; but after a few hours, the course of the disease will clear the diagnosis. When the lesions remain untreated, they become irregular in character and widespread in distribution; so that after a day or two, especially in older infants, they may be seen here and there over widely separated parts of the body, entirely lacking that continuity so characteristic of erysipelas in adult patients.

If the face is the site of the erysipelas, toxic edema in the loose areolar tissues is a feature, and the eyelids become so swollen that they cannot be opened. On the scalp, the swelling may become extreme and thrombosis of the emissary veins may take place and intracranial involvement follow. In the newborn, an erysipelas of the body may advance and involve the umbilical wound and in this way the streptococcus invades the peritoneum and the blood stream, with a fatal issue.

The *treatment* of erysipelas in infancy is thoroughly satisfactory. If it can be applied promptly before the lesions have become widespread, the disease may often be checked within 4 or 5 days. Leucocytic extract acts most effectively, but it must be given in large doses frequently repeated. The preparation that has proved the most useful in our hands is the so-called "noninflammatory extract," made from equine leucocytes separated from the blood by centrifugal action and then digested. With one exception, we have observed no untoward reaction follow the use of this preparation. Two c.c. of the leucocytic ex-

tract (adult strength) are injected even into the smallest babies. The preferred site for injection is the triceps muscle and the dose may be repeated at 8- to 12-hour intervals until the symptoms abate.

For local application, there is nothing that in any way approaches ichthyol for combating the skin manifestations of the disease. However, it must be used in a vehicle which will penetrate the skin. Applied in watery solution or combined with mineral oils, it is practically useless, but used in a 25 per cent ointment with a lard base, it is decidedly effective. Iced compresses of boric acid may be used on the eyelids when they are swollen. The frequently advocated compresses of magnesium sulphate or aluminum acetate have proved fruitless in our experience. Magnesium sulphate may be irritating to the infant's skin and for this reason its use is to be avoided. Helmholz has accomplished the cure of erysipelas in babies by 3 or 4 periods of exposure of the affected area—50 centimeters from the quartz lamp, for 2 minutes, increasing ½ to 1 minute each day, depending upon the amount of reaction.

Erysipelatous children sometimes also suffer from cardiac weakness and may need stimulation with caffein sodium benzoate; ¼ grain to ½ grain doses are usually effective. When heart failure is extreme, it may be necessary to use camphor in oil, strophanthus or digitalis.

Pyrexia or hyperpyrexia is to be met by the use of hydrotherapy. The nutrition must be maintained. Care should be taken that the food is not overconcentrated or the meals too frequent. Children with erysipelas require a full quota of fluid which is well given in the form of fruit-juices diluted with water. The addition of the mild alkalies is sometimes of advantage. The citrate of potassium is palatable and it is readily taken.

Scarlet Fever

Like the other acute infectious diseases, **scarlatina** is a disease that occurs more frequently through the school age than it does in infancy. During the first year of life, it is rare except as a part of a house epidemic; while it is of a little more frequent occurrence the second year of life, it is relatively uncommon during this period. Fortunately, it is the **mild type** rather than the **malignant** that is met at this age, although in later infancy, the **anginal** form may be seen.

The onset of the disease is almost invariably abrupt and is ac-

companied by vomiting and by an increased pulse rate. While the rash usually appears within 48 hours, rarely, it may be delayed for 5 or 6 days. It is characteristic in appearance and in distribution. Before the rash develops, and during the first day or two of its manifestations, flushing of the cheeks with circumoral pallor and pinched white nose may be seen. This facial phenomenon is rarely present in any other disease than scarlet fever. After 48 hours, there is a fine almost imperceptible desquamation over the cheeks which causes an appearance not unlike that produced by the application of rouge with a little white powder dabbed over it.

At this time, the true rash, which appears first on the chest, spreads until it includes all of the body except the face. Rarely it may run up onto the jaws and back of the ears. The typical picture of this eruption is produced by the fact that there are two elements in its make-up; one a general erythema, and the other a punctate reddening, the puncta blending into the erythema. The rash may be so slight as to escape attention, or it may be of the greatest intensity and the skin studded with petechiæ. In malignant cases, these petechiæ are widespread, and from the onset the rash may be purplish rather than scarlet in color. This peculiarity of hue is the result of a marked cyanosis of the skin. The nose, ears and lips will be extremely blue and there may be a general waxy pallor, like that seen in other generalized septic infections. Children with this type of rash rarely live more than 2 or 3 days.

The mouth, tongue and fauces present an appearance which varies with the stage and intensity of the disease. At the beginning, the tongue papillæ are enlarged and red and they protrude as reddened dots through the white, sodden surface of proliferating epithelium. This tongue is described by Stephen McKenzie as the "strawberry and cream" tongue; this is in contradistinction to the raw tongue surface with its enlarged papillæ which appears after a few days and which has been descriptively called the "raspberry tongue".

In mild cases, the pharynx is always inflamed and there is a reddening of the faucial pillars which disappears after a few days. In the anginal forms, we may have any involvement from an intense reddening of the tonsils and pillars with swelling, to a process severe enough to produce sloughing. Such a slough may lead to perforation of the pillars or even to a deep ulcera-

tion with severe hemorrhage. In certain cases in which the anginal form with ulceration is pronounced, or when there is reason to suspect a complicating streptococcic septicemia, the appearance of the usual scarlatinal rash may be modified. On the third or fourth day, a dull red maculo-papular eruption appears which is quite distinct from the previous typical rash of the disease. The distribution of this secondary rash is limited to the extensor surfaces of the arms and legs; rarely, it reaches the body and face.

Together with the faucial symptoms just mentioned, there will be a marked rhinitis both anterior and posterior, with an irritating discharge from the nose, sometimes watery, sometimes purulent. The pathological process here may cause destruction of the tissues of the nose, ulceration of the soft palate and even laryngeal inflammation with or without ulceration. In this anginal form of the disease, which is probably merely the evidence of a complicating streptococcus infection of the nose and throat, a cervical adenitis is almost invariably part of the picture. When the faucial symptoms are slight, the adenitis is of low degree; although even with moderate swelling, suppuration of one or more of the cervical glands may occur and incision with evacuation of pus may be needed. In many cases there is a great deal of purulent or semipurulent discharge from the nose or from about the fauces. When the ulceration in the throat is profound, the involvement of the lymphatics and the deeper tissues of the neck may take place and result in the brawny swelling with induration in the cervical region known as **Ludwig's angina**. This condition is almost invariably fatal. Malignant cases of scarlet fever are so rapidly fatal that there is rarely time for the development of much faucial complication.

Little children are subject to other eruptions of the skin which are not unlike the rash of scarlet fever, but which are self-limited and pass off within a few hours or days at most. However, these are unaccompanied by any faucial signs. Such erythemas appear on the face as well as on the body. The same is true of the **toxic erythemas** which are known to follow the use of soapsuds enemas and the ingestion of various drugs, notably, quinin, belladonna and antipyrin.

There is a form of **erythema, (erythema scarlatiniforme)**, which resembles scarlet fever but which probably is closely related to urticaria. It is noncontagious and usually results as a complica-

tion of some other infectious disease or as a sequela of a food sensitization. Except for its cutaneous manifestations, it has no similarity to scarlet fever. This exfoliative dermatitis is not a disease of early infancy. There are, however, in the newborn, cases of **desquamative erythema**, the result of septic infections; in these, the source of the erythema is so clear that a mistake is not apt to be made. The serum rashes may give a skin picture closely simulating the ordinary manifestations of scarlet fever, but with the history of the injection of a foreign protein at hand, it is not likely that the observer will be led into error.

The blood in scarlet fever has very little diagnostic significance. There is a leucocytosis of varying degree in practically all cases, no matter of what type. This leucocytosis of the polymorphonuclear variety begins early in the disease and, as a rule, reaches its maximum during the first week. The more severe the case, the higher the leucocytosis; and of course pyogenic complications have their usual effect on the curve. In a certain percentage of the patients, there is a moderate eosinophilia which appears and persists during the first part of the illness. A return of the eosinophilic percentage to normal has been noted to be of good prognostic omen.

The **scarlatiniform** type of eruption in **German measles** may present a diagnostic problem difficult to elucidate. There is nothing more taxing than to make a definite differentiation between a mild scarlatina and a scarlatiniform rubella. The history of contact together with the long incubation period; and the entire absence of anginal symptoms; the lymphocytosis; the disappearance of the fever coincident with the appearance of the rash; and the early enlargement of the occipital lymph nodes, all seen in rubella, are points that will aid in reaching a decision.

Vomiting is a usual initial symptom of scarlet fever even in the milder cases. In the more severe cases, the emesis may be protracted and taxing; in the toxic group, the vomiting is often extreme, unmanageable and almost continuous.

Pyrexia is a constant phenomenon of the disease, but in some of the milder cases it is so transient that it may be easily overlooked. The fever, which usually ranges from 102° to 104°, begins abruptly, maintains a fairly flat curve for some days and drops by lysis. In the anginal cases, the temperature curve will be influenced by the amount of ulceration and infection present in the throat. In the fatal toxic cases, there is usually a hyperpyrexia, but occasionally the disease may run an apyrexial course,

especially when there is much hemorrhage. The usual teaching that the pulse rate is out of proportion to the temperature is difficult to prove in the case of infants whose pulse rate is well known to be variable.

The *treatment* of scarlet fever contemplates such measures as will apply to any patient suffering from an acute infectious disease. The child should be put to bed in a sunny, well ventilated room, isolated from other members of the family. In the mild cases, no particular treatment is needed other than the provision of general hygienic measures. The diet should be easily digestible and given in sufficient quantity to provide for the child's nutritive needs. There should be a careful toilet of the mouth, nose and fauces. For this purpose, nothing is better than a 2 per cent sodium bicarbonate solution sprayed warm from an atomizer; if preferred, dilute alkaline antiseptic or boric acid solution may be used. The bowels should be kept open, preferably by mild laxatives, as these are more effective than colonic flushings in producing watery evacuations. The passage of water by the bowel relieves the kidneys of a certain amount of work. Milk of magnesia is a useful mild laxative, but it must be given in sufficient dosage, at least 2 teaspoonfuls during a day to a 20-pound baby.

In most cases, the fever is readily controlled by proper sponging. There is no objection to the use of immersion baths; when hyperpyrexia is a feature, tepid packs are of great value. When either toxic or cardiac complications develop, the use of the mustard pack should be resorted to. (See Methods.)

In the stage of desquamation, inunctions of simple unguents are comforting. For the itching of the skin, alkaline baths often give relief, and the daily use of the cresol bath in the proportion of 1 dram to the gallon hastens the process of desquamation, in addition to allaying cutaneous irritation.

Measurement of the 24-hour urinary output is an aid to the intelligent treatment of the disease and is particularly helpful after the first 5 or 6 days. A diminution of the amount of urine excreted is the first sign of kidney involvement; should this symptom appear, immediate treatment of the urinary complication must be instituted. In young children, it is sometimes difficult to collect the urine, but this difficulty can be overcome (see Methods).

In the anginal forms of scarlet fever, lavage of the nose and

the throat with mild, warm alkaline solutions should be a routine part of the treatment. Many cases of suppurating and ulcerative anginas can be prevented if early and sufficient irrigation of the upper respiratory tract is practiced. There is no more reason why a sloughing, necrotic wound of the fauces should not receive scrupulous care than there is for neglect of a wound in any other part of the body. Lavage properly applied (see chapter on Methods) will tend to keep the affected area free of discharges and in this way prevent the development of adenitis and cellulitis. Any of the mild alkaline solutions, such as borax solution, alkaline antiseptic solution or sodium bicarbonate solution, are to be preferred to the solutions such as Dobell's; for the cleansing process is entirely mechanical, and stronger solutions only add to the irritation without having any decided bactericidal effect.

Local applications to the throat are of value, but they should be applied only by the physician. The frequent touching of the fauces with mild antiseptics does little good and succeeds only in making a sick child very unhappy. A silver nitrate solution, 15 per cent to 25 per cent, is very effective; or iodine, 10 per cent, dissolved in glycerin may be used if preferred. Iodin crystals, 5 per cent; iodide of zinc, 2 per cent, in glycerin and water equal parts, make a potent mixture for local application. Great care must be taken in applying these solutions, for if an excess is allowed to run down into the glottis or the larynx it may do much harm.

In cases of adenitis in which there is much swelling, an ice collar often affords great relief but it should not be continuously applied. The ice should be finely shaved, as large lumps are uncomfortable. Older children should wear it during alternate hours; younger children will not tolerate applications of this length, and they should wear the collar for not more than half an hour at a time. Failing an ice bag, cold compresses frequently changed may be used. If there is indication of approaching suppuration in any of the glands of the neck, hot applications, because they hasten softening, are preferable to cold. When liquefaction has fully developed, evacuation should be made through the smallest effective incision; it is surprising how small an opening, if it be kept open, will allow the free escape of pus. Many a huge slough has resulted from too wide incision in cases where the tissues resistance is low.

The treatment of otitis media differs in no way from treatment

of ear infections due to any other cause. A daily inspection of the ears of infants who suffer from acute infectious diseases is always in order, and at the first appearance of a bulging drum, paracentesis of the tympanic membrane should be made without delay. This should be followed by daily cleansing of the aural canal with cotton swabs or by mild antiseptic irrigations through a soft rubber aural syringe (see Methods).

In toxic cases of scarlet fever, eliminative measures are indicated. The administration of saline purges sufficient to produce several large watery evacuations each day, is an efficient means to this end. The kidneys should be stimulated by administration of water, by mouth if the patient is able to swallow, or given intraperitoneally, subcutaneously or intravenously. It is advisable to add sugar to the water unless it is given subcutaneously. It may be given as glucose solution 4½ per cent intravenously, in the form of lactose solution, 5 per cent to 8 per cent, by mouth, or as a hypertonic glucose solution, 10 per cent, by proctoclysis.

Lethargy and somnolence are often most marked in the extremely toxic cases; these symptoms can be combated best by the use of the mustard pack. This procedure stimulates the sensorium, produces rapid diaphoresis and it may have some value as an aid to elimination (see Methods).

At the very earliest indication of cardiac weakness, stimulation becomes necessary. In an emergency, the intravenous administration of strophanthin, 1/1500 of a grain for a 20-pound infant, is very effective. This may be followed by the slower acting heart stimulants such as an alkaloid of digitalis given hypodermically or digitalis by mouth in the form of a fresh infusion or of an approved tincture. If preferred, caffein, ⅛ to ½ grain, or camphor, 10 per cent in oil, 8 to 10 minims, may be used by hypodermic needle.

In those cases of scarlet fever which exhibit profound symptoms from the onset of the disease and which run a rapid and alarming course, the intravenous injection of a polyvalent streptococcus serum may prove of value. A small dose of the serum should be given half an hour before the therapeutic injection in order to desensitize the patient and thus to avoid the distressing anaphylactic phenomena which sometimes occur. The serum should be well diluted with normal salt solution and be given slowly into the longitudinal sinus, or into the external jugular vein in those infants in whom the fontanel is closed (see Methods).

Acute nephritis complicating scarlet fever is of two types; the one, which overwhelms the kidney early, and the other, which appears from 3 to 5 weeks after the disappearance of the rash. In the first, the urine shows red blood cells, blood casts and white blood cells; it is scanty, highly colored and concentrated. As these urinary changes may be the only signs discoverable for some time, unless careful routine examination be made, the patient may suddenly surprise the physician by developing uremia. The late oncoming second type of scarlatinal nephritis first shows itself by a puffiness of the eyes and edema of the face, hands, feet and body, and sometimes even by a general anasarca. The urine then becomes that of a glomerular nephritis; it contains albumin, blood, blood-casts, and pus.

The treatment of this complication differs in no way from the treatment of a glomerular nephritis of any other etiology and is discussed in detail in the chapter on Genitourinary diseases.

As a sequela of scarlet fever in older infants, "scarlatinal rheumatism" may appear. The tissues involved are those of the joints, the synovia of the tendons, especially the extensor sheaths of the tendons of the wrist and those running over the dorsum of the foot. The process is self-limited and, except for the fact that it is painful, is of little importance. Sometimes the pericardium, myocardium or endocardium become involved with a subsequent crippling of the heart—an accident quite unlikely to occur during the first 2 years of life. The painful areas about the joints are relieved by the application of fomentations. Fixation by splint provides added comfort. The salicylates seem to hasten recovery and to diminish pain in some cases, but they very often contribute to emesis and therefore should be used with caution.

Thorough isolation should be rigidly enforced and the strictest measures taken to prevent the spread of the infection. No one should be allowed in the sick room except the patient, the attendant and the physician. The nurse should wear rubber gloves when handling the patient, and a 1 per cent cresol solution should always be kept conveniently near for the purpose of immersing the gloved hands. When soiled, all clothing and bed linen should be dropped in a large container holding 1 per cent cresol and they should be boiled in this solution before being laundered. Utensils and dishes used by both patient and nurse should be boiled in the sick room if possible, before being sent back to the family

kitchen. The attendant should wear a washable slip-over, which when she leaves the room for her daily exercise and airings should be removed and replaced by one that has not been subjected to contamination. While out of doors, the nurse should take particular pains to allow no one to come near her. Health regulations in various communities differ in the matter of the term of quarantine; but in general it can be stated that it is unsafe to permit the patient to come in contact with people until at least 2 or 3 weeks afer the cessation of all symptoms, and not then unless all discharges from the nose and throat or ears have disappeared.

When quarantine is raised, both patient and nurse should have a thorough bath and shampoo before coming in contact with the outside world. Soap and water are sufficient for this purpose although there is no objection to a cresol bath unless the patient's skin is irritated and tender. Complete changes of clothing should be provided both nurse and patient. The sick room should be left vacant for several days while it is thoroughly aired. Afterward, the room should be given a complete cleaning and all its contents should be exposed to the sun for several days.

PART III

CHAPTER XXII

METHODS

The methods of procedure given in this section are all useful in dealing with sick children. The manner of doing any given thing naturally varies with the predilection of the doer. The writers have found that by arranging and writing out the procedures, step by step, that they have facilitated and simplified the work for themselves, their associates and assistants. There are, doubtless, many other ways of accomplishing the same ends and many of them are probably equally good. The schemes of procedure as given here are those that have stood the tests of practice in our hands, and we are confident in recommending them to others.

Any of the procedures which demand breaking of the skin, call for strict aseptic precautions which have not been insisted upon in each instance, because it is assumed that the reader needs no particular instruction in the principles of asepsis.

INTRAVENOUS INJECTION

Intravenous injection is the most rapid, direct and effective method of administering fluids in large amounts for therapeutic purposes.

The **fluids** available for injection by this method are:

1. Normal salt solution, (sodium chloride, 0.8% in distilled water).
2. Glucose solution, (glucose, 5 % to 20% in normal salt solution).
3. Acacia solution, (gum acacia, 7%, glucose 5% in normal salt solution).
4. Fischer's solution, (sodium carbonate, 2%, sodium chloride, 1.4% in distilled water).
5. Citrated blood.
6. Serums, (such as antimeningococcic, in normal saline solution).

7. Medicaments, (such as arsphenamin solutions).

Of these, all are of value in appropriate cases, with the possible exception of Fischer's solution. This formula has been much used as a therapeutic measure to combat acidosis, but it has little or no advantage in infants over the injection of an equal amount of normal saline solution.

Equipment.—1. An intravenous needle, size 18. The needle should be short-beveled, the point should be sharp and not bent; it is important that there be no rust on the needle, either inside or outside. If the longitudinal sinus is to be used, the beveled block needle devised by Goldbloom of Montreal is the needle of choice. (Fig. 1.)

This mechanism is composed of "a needle 4 cm. long with obturator, fitted into a rectangular block 3 centimeters in thickness, thereby allowing 1 cm. of the needle to project." Goldbloom has modified the original apparatus by beveling off the block at an angle of 50°, so that the needle enters the vein at such an angle rather than at right angles. The block contains a set-screw so that the part of the needle that projects through the block may be fixed at any desired depth.

2. A metal connector of appropriate size, so that it will accurately fit into the base of the needle. If the needle be of the "slip-connection" type, the connector will not be necessary.

3. Rubber tubing, ⅛ inch in diameter and 1 foot long. The tubing should be resilient enough not to collapse or buckle at the connections. The metal connector should be fitted into the end of this tubing.

4. Glass connector of the same size as the tubing.

5. Rubber tubing, ⅛ inch in diameter and 2 feet long. The long and the short pieces of rubber tubing should be connected with the glass connector.

6. Container. This should be graduated and of glass—preferably a Kelly flask. In emergency, a metal container may be used.

7. A solution thermometer. A fever thermometer will serve the purpose unless it is desired to give solutions above 110°.

8. Hot water bag for suspension outside the container to keep the fluid at a constant temperature.

9. Tourniquet of soft rubber, 1/16 or ⅛ inch in diameter (if the vein at the bend of the elbow is used).

10. Sterile towels.

11. Green soap solution.

12. Iodin solution, 2%.
13. Alcohol, 50%.
14. Sterile gauze, sponges and dressings.
15. Scalpel, thumb-forceps, probe, hemostats, needles, small hypodermic, 1% novocaine solution and catgut (in case it becomes necessary to expose the vein, if the injection is being made at the bend of the elbow. This procedure is seldom or never necessary).

The Site of Injection

A. Superior Longitudinal Sinus.—

This is a favorite site for injection of fluid into the circulation in infants whose anterior fontanel has not yet closed. The superior longitudinal sinus traverses the diamond-shaped area that is known as the anterior fontanel, from its anterior apex to

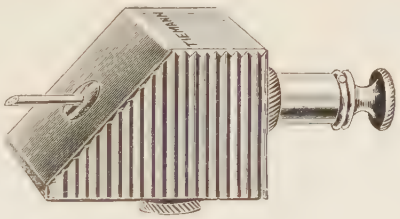


Fig. 1.—Goldbloom needle.

its posterior apex. It increases in diameter posteriorly. It lies but $\frac{1}{16}$ to $\frac{1}{8}$ inch beneath the skin and it is easily accessible.

Procedure.—

(a) Wrap the patient in a sheet, restraining the arms to the sides, pin in snugly and lay him on his back on a table. The top of the head should extend just over the margin of the table. Have an assistant hold the head firmly (Fig. 2).

(b) Shave the hair off the area of the anterior fontanel.

(c) Cleanse area with green soap solution, wash off with alcohol, dry thoroughly, apply 1% or 2% iodin solution, remove the iodin stain with alcohol.

(d) Drape the area with sterile towels, or better, use a specially prepared scalp-sheet about 18 inches square, with a hole 2 inches in diameter in the center.

(e) Inspect apparatus, test connections and if the Goldbloom needle is used, set the needle at the required depth. In young and very thin babies, the needle should project from the block about $\frac{1}{16}$ inch; in older infants, about $\frac{1}{8}$ inch.

(f) Exhaust the air from the rubber tubing by allowing normal salt solution to flow through the needle for a few moments. Be certain that all the air has been forced out.

(g) Test the temperature of the fluid with a sterile thermometer. If it is desired to administer fluid at body temperature, the fluid in the container must be maintained at 101°, because at this level, it loses about 2° in transit, (assuming a room temperature of about 75°). If it is desired to give fluids at 110° or above, the temperature in the container must be at least 10° higher, as the radiation of heat through the container and the tube in such an instance is much faster. A hot water bag, (suspended against the container and held in apposition by a towel,

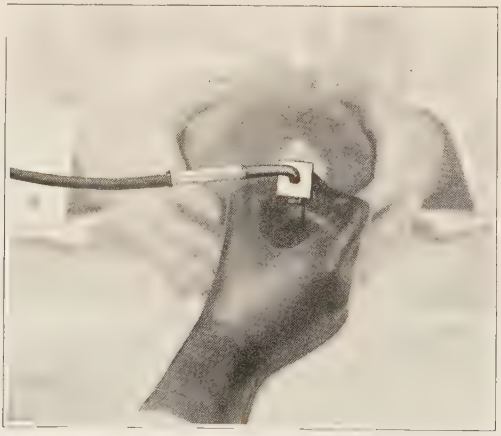


Fig. 2.—Injection into longitudinal sinus.

pinned top and bottom, leaving the graduations visible), is a convenient means of maintaining a constant temperature of the fluid in the container.

It is very important that the fluid should be gravitated into the vein of an infant at body temperature or above. Many infants to whom this therapeutic measure is helpful, have subnormal temperatures and chilling is often productive of grave consequence.

(h) Locate the apex of the posterior angle of the fontanel with the index finger of the right hand; insert the needle here pointing it backwards at an angle of about 50°, to a depth of $\frac{1}{16}$ to $\frac{1}{8}$ inch. Remove the obturator and if it is followed by a flow of blood, the sinus has been entered. If no blood appears, re-

turn the obturator and insert the needle a little further. If the Goldbloom needle is used, greater depth may be attained by loosening the setscrew. Rarely, the sinus may be missed entirely at the posterior angle of the fontanel. The writers have encountered such an instance a few times, when they were dealing with infants who were rachitic and whose skulls had developed asymmetrically. In such a case, a clear cerebrospinal fluid will emerge from the needle, but this need cause no alarm. Other attempts may be made by inserting the needle at one or the other side of the median line.

(i) Once the sinus is entered, the connector on the rubber tubing should be inserted into the base of the needle *while the fluid is flowing.* This expedient prevents the injection of air into the vein.

(j) The rate of flow of the fluid into the vein may be controlled either by raising or lowering the container, or by maintaining moderate pressure on the tube with the thumb and forefinger. At least 10 minutes' time should elapse while 150 to 200 c.c. of fluid is being given.

(k) When the required amount of fluid has flowed into the sinus, withdraw the needle sharply and make firm pressure over the stab wound with a gauze sponge. After a few moments, apply a collodion dressing. The dressing should be removed in 24 hours, as sealed wounds may suppurate.

B. External Jugular Vein.—

The site of the external jugular vein is the second of choice for intravenous injection in infants. That portion of the vein between the sternomastoid muscle and the clavicle is fairly accessible except in very fat infants. For equipment for this operation, see page 482.

The Procedure.—

(a) Wrap the patient in a sheet, restraining the arms to the sides and pin the sheet snugly.

(b) Lay the patient on his side on the table (Fig. 3).

(c) Have an assistant support the head and pull the upper shoulder downward, thus throwing the posterior triangle of the neck into prominence.

(d) Cleanse the area with green soap solution, wash off with alcohol, dry thoroughly, and apply 1% to 2% iodin solution, remove the iodin stain with alcohol.

(e) Suspend the container about 2 feet above the level of the

site of injection. Inspect apparatus, test connections and have temperature of fluid taken at the outlet of the tube by running 50 c.c. or 100 c.c. of the fluid into a warm glass containing a thermometer. The temperature should be at least 101° in the container and so maintained throughout the procedure.

(f) Exhaust the air from the rubber tubing by allowing normal salt solution to flow for a few moments. Be certain that all the air has been forced out.

(g) Choose the most prominent portion of the vein posterior to the sternomastoid muscle. A little pressure just above the

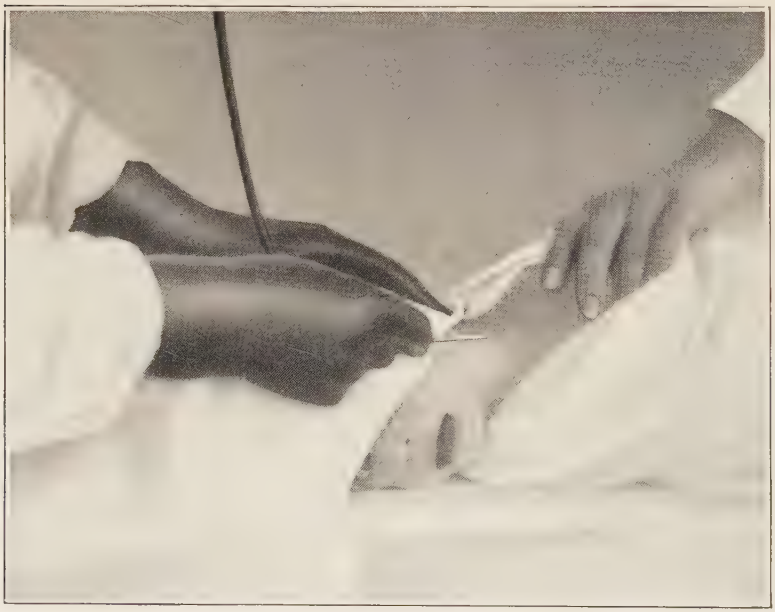


Fig. 3.—Injection into external jugular vein.

clavicle may make the vein more visible. Insert the needle with one motion through the skin and into the vein. Do not pick up the skin with the thumb and forefinger and insert the needle as in giving a hypodermic, and then try for the vein. If the bevel of the needle is held towards the surface of the skin, there is less chance of transfixing the vein. The needle should be inserted almost parallel with the course of the vein and should point toward the clavicle. The appearance of blood at the base of the needle marks successful entry.

(h) Connect the tubing to the base of the needle *while the*

fluid is flowing. This maneuver prevents the entry of air into the vein.

(i) Control the rate of flow either by lowering or raising the container or by making moderate pressure on the tube with the thumb and index finger. Mechanical clips are not satisfactory. At least 10 minutes should elapse while 100 c.c. to 200 c.c. are being given.

(j) When the required dosage has been given, withdraw the needle sharply, make firm pressure over the wound for a few moments, and apply a sterile dressing, holding it in position with adhesive plaster.

C. At the Bend of the Elbow.—

The median-basilic or the median-cephalic vein is sometimes chosen for intravenous injections in infants, but it is often very difficult to enter the vein at this point unless the vein is exposed, especially in babies who have a considerable amount of subcutaneous fat. For the equipment required for this operation, see page 482.

The Procedure.—

(a) Wrap the patient in a sheet with the chosen arm excluded. The sheet must be snugly pinned to obviate struggling.

(b) Place the patient on his back with the arm extended on the table and the forearm in supination. Have an assistant comfortably seated to hold arm in this position.

(c) Put tourniquet loosely in position around the upper third of the arm.

(d) Cleanse the area with green soap solution, wash off with 50% alcohol, thoroughly dry, apply 1% or 2% iodin solution, remove iodin stain with 50% alcohol.

(e) Drape body and arm with sterile towels, or better with a specially prepared sheet with a 2 inch hole in the center.

(f) Tighten tourniquet. Great care must be taken not to tighten the rubber tube tourniquet more than just enough to hinder the venous blood flow. It is surprising how slight a degree of pressure is necessary to accomplish this purpose in an infant. Too great pressure applied through the tourniquet will interfere with the arterial flow and as a result, a collapsed vein may render the operation impossible.

(g) Suspend the container about 2 feet above the level of the site of injection. Fill with normal salt solution at the desired temperature, which must be maintained throughout, test the con-

nections, allow the fluid to flow and test the temperature at the outlet by allowing a few cubic centimeters to run into a warm glass containing a thermometer. The temperature of the fluid should never be below body temperature. If apparatus is satisfactory, have assistant stop the flow by pinching off the tube.

(h) When the vein becomes prominent, hold the point of the needle against the skin over the vein with the needle's bevel toward the surface. Insert the needle into the vein with one motion. Do not pick up the skin with the thumb and forefinger as in giving a hypodermic, and then try for the vein. If the vein is not entered the first trial, then the searching process may begin.

(i) In case it is impossible to enter the vein, it will be necessary to expose it. If this procedure is decided upon, infiltrate the skin over the vein with a 1% novocain solution, using a hypodermic with a needle of small caliber. Make a one-half or three-quarter inch incision through the skin, carefully dissect out the distended vein, insert a small probe under it, throw a ligature about its proximal end without tying, ligate off the distal end, insert the needle into the lumen of the vein and fasten it in position with the untied ligature above mentioned.

(j) When once the flow of blood through the needle has been established, loosen the tourniquet, start the fluid flowing through the rubber tubing and make the connection.

(k) Control the rate of flow by raising or lowering the container or by making pressure on the tubing with thumb and forefinger. Allow at least 10 minutes time to elapse while giving 100 c.c. to 200 c.c. of fluid.

(l) When the required amount of fluid has flowed into the vein, remove the needle sharply, suture skin wound, apply sterile dressing and fasten into place with adhesive plaster.

BLOOD TRANSFUSION

The direct method is preferable. Injection of citrated blood (described herewith) may be mandatory in emergency.

Choice of Donor.—

1. The father or mother of the patient.

2. Other persons. When a parent is not available, the clinical evidence of syphilis should be searched for and a Wassermann test should be made.

Grouping.—

Relationship of the donor does not insure against hemolysis. The hemolytic reaction between the serum and corpuscles of the donor should be tested against those of the patient. The reactions are such that four groups of blood are known. One group, called Group 4, is universally utilizable but it is better that the blood of both donor and recipient should be of the same group. The method of grouping is based upon the agglutination of the recipient's corpuscles by the donor's serum. The technic of grouping is a delicate procedure, and it should be left to a trained laboratory worker.

Choice of Site of Injection.—

1. Longitudinal sinus.
2. External jugular vein.
3. Median-basilic or median-cephalic vein.

Equipment for Collection of Blood.—

1. Intravenous needle of "slip connector" type, size 16 or 17 with 3 inches of ⅛ inch rubber tubing attached.
2. Rubber tubing, ⅛ or ¼ inch for tourniquet.
3. Cylindrical graduate or a wide-mouthed bottle of 200 c.c. capacity.
4. Glass stirring rod.
5. Sodium citrate solution, 2%; 10 c.c. for each 100 c.c. of blood to be collected.
6. Green soap.
7. Alcohol, 50%.
8. Iodin, 2%.
9. Gauze, dressings and adhesive plaster.

Equipment for Injection.—

1. The equipment for intravenous injection, page 482.
2. A filter composed of several layers of fine gauze with the cut margins infolded and sewed in such a manner that small threads may not go on through the filter with the filtrate.

Position of Recipient.—

1. Wrap patient in a sheet restraining arms, lay on his back on the operating table. The top of the head should come just over the margin of the table.
2. Have patient's head firmly held by an assistant.

Position of Donor.—

1. Seated in a chair, preferably in adjoining room, or
2. On an operating table.

Preparation of Donor.—

1. Cleanse the skin at the bend of the elbow with green soap, dry with alcohol.
2. Apply 2% or 3% iodin solution.
3. Remove iodin stain with alcohol.
4. Cover area with sterile towel.

Preparation of Recipient.—

1. Shave scalp over anterior fontanel if the longitudinal sinus be chosen.
2. Cleanse with green soap, dry with alcohol.
3. Apply iodin solution, 1%.
4. Remove iodin stain with alcohol.

Collection and Citration of Blood by Assistant.—

1. Place in container 10 c.c. of 2% sodium citrate solution for every 100 c.c. of blood to be collected.
2. Tighten tourniquet on upper ⅓ of the arm, just enough to interfere with the venous flow of blood.
3. Make the vein puncture, pointing the needle toward the hand.
4. Collect the blood in the container while the tourniquet is still in position.
5. Stir gently but constantly while the blood flows into the citrate solution, to insure thorough mixing.
6. Release tourniquet, withdraw needle and apply dressing.

Injection into Recipient.—

1. Place 200 c.c. of salt solution into container and exhaust the air from the tube by allowing the salt solution to flow through the needle.
2. Add the citrated blood to the container while there are still a few c.c. of salt solution visible. The citrated blood should be passed into the container through a funnel containing the gauze filter.
3. If the longitudinal sinus is chosen, locate the posterior angle of the anterior fontanel, insert the needle at the apex pointing it backward at an angle of 50°. When blood appears at the base of the needle, connect the tubing by inserting the metal connector into the base of the needle while the fluid is flowing.
4. Control the rate of flow by raising or lowering the container or by making pressure on the tube with thumb and forefinger. Allow at least 10 minutes to elapse while 200 c.c. are being given.
5. Allow 100 to 300 c.c. of citrated blood to enter the vein.

6. Remove the needle, make slight pressure with a piece of gauze and apply collodion dressing.

7. Remove the dressing in 24 hours.

(NOTE: If it is desired to use the external jugular vein or the veins at the bend of the elbow, the technic is the same as for giving fluid intravenously).

INTRAVENOUS INJECTION OF ARSPHENAMIN (SALVARSAN, ARSENOBENZOL)

Note.—Seldom employed in young children. Neoarsphenamine drug of choice.

Site of Injection.—(Never into longitudinal sinus.)

The median basilic, the external jugular or the veins of the scalp.

The Equipment.—

1. Ampoule of arsphenamin and a small file.
2. *Sterile, freshly distilled* water, 300 c.c.
3. A mixing chamber with ground glass stopper.
4. Two 500 c.c. graduates.
5. Glass stirring rod.
6. Glass funnel.
7. Filter paper, white, washed and sterile.
8. Medicine dropper.
9. Freshly prepared sodium hydrate, 15 per cent.
10. An intravenous outfit, see page 484.

Position of the Patient.—

1. If the veins at the bend of the elbow are chosen, wrap child in sheet restraining one arm and leaving the other free; lay the patient on his back on the table with the free arm extended from the body and the forearm in supination.

2. If the external jugular vein is chosen, wrap child in a sheet restraining its arms to the side; lay on its side on the table with head deflected to one side and the uppermost shoulder depressed.

The Procedure.—

1. If the vein at the bend of the elbow be chosen, lay the patient on his back with the forearm extended and the arm at right angles to the body. Put the tourniquet (rubber catheter) into place.

2. Cleanse the area with green soap solution and dry with alcohol.

3. Apply 2% iodin solution.

4. Remove iodin stain with alcohol.

5. Drape the area with towels, or use a "scalp-sheet" (a square piece of sterile muslin with a 2 inch hole in the center).

6. Lift the ampoule of arsphenamin out of the alcohol solution, dry with sterile gauze.

7. Make a transverse file mark across the shoulder of the ampoule and break off the neck.

8. Pour 30 to 40 c.c. of *sterile, freshly distilled* water *at room temperature* into the mixing chamber; this is the appropriate amount of water for 0.5 grams of arsphenamin.

9. Sprinkle drug on the surface and agitate. It will go into solution without difficulty. After solution is clear,

10. Add sodium hydrate, 15 per cent and freshly prepared, drop by drop from a medicine dropper. (About 20 drops are required to neutralize each 0.5 grams of arsphenamin.) A flocculent precipitate forms; this precipitate disappears on shaking the solution.

11. Add sterile saline solution *at room temperature* (made with C. P. sodium chloride, 0.5 per cent with freshly distilled water) to bring the total volume of fluid up to 250 c.c.

12. If the solution is not clear, add a very few drops more of the sodium hydrate solution; wait two or three minutes and repeat if necessary.

13. Filter if there is any chance of there being any extraneous matter such as broken glass in the solution.

14. Put about 200 c.c. of normal salt solution into the container of the intravenous outfit. Allow the salt solution to flow and exhaust the air from the tube. When all the air has been forced out of the tube, pinch it off leaving 50 c.c. or 100 c.c. of normal salt solution in the container.

15. Tighten the tourniquet moderately and fasten with a single loop knot in order that it may be easily removed when the vein is entered. Allow arm to hang over the edge of the table for a few minutes, then attempt to palpate the vein. Insert the needle, bevel down, through the skin and into the vein with a single motion if possible rather than to pierce the skin first and then to try for the vein.

16. As soon as the blood flows through the needle, loosen the tourniquet, connect the rubber tubing and allow the normal salt solution to flow from the container into the vein.

17. Watch the level of the fluid in the container by comparing it with the graduation marks, and as soon as it is evident that the fluid is flowing well, break the connection and allow the normal salt solution to escape into the waste pail. When the level of the salt solution in the container has just reached the bottom, have an assistant pour the arsphenamin solution into the container.

18. Allow the arsphenamin solution to gravitate into the vein. Control the rate of flow by raising or lowering the container. At least 10 minutes' time should elapse while the dose is being given.

19. Just as the last of the arsphenamin solution disappears from the container into the tube, have an assistant pour 100 c.c. of normal salt solution into the container. This last maneuver will insure that the patient gets the full dose.

20. When the color of the fluid passing through the glass connector indicates that normal salt solution is flowing, sharply withdraw the needle, make pressure over the wound for a few moments, and apply collodion dressing.

(NOTE: It is important when the apparatus is being sterilized by boiling, in tap water, that all glass containers should be individually wrapped in towels. This simple expedient prevents the collection of alkali on the glassware from the boiling water. These precipitates, particularly if the water is very "hard" often interfere with the neutralization process. Sterilization in distilled water eliminates this difficulty.)

Never use hot water in making up solutions of arsphenamin.

If the external jugular vein is chosen for injection, the technic differs from that just described only in the method of entering the needle into the vein.

INTRAVENOUS INJECTION OF NEOARSPHENAMIN (NEOSALVARSAN, NEOARSENOBENZOL)

This form of arsenic does not require the neutralization process. The drug goes into solution with great ease. One decigram will dissolve without difficulty in 1 c.c. of water. The drug may be given at any of the sites applicable for the injection of arsphenamin. In addition to the advantage of not requiring neutralization, the solution may be injected intravenously, in concentration, with a small glass syringe. While the intravenous

outfit may be used, it is not necessary. A glass syringe of from 1 c.c. to 5 c.c capacity, preferably with a Becton-Dickinson connector, and a needle 25 gauge if the scalp veins are used, are all that are required. The solution may be mixed in the barrel of the syringes by removing the piston, pouring freshly-distilled water at room temperature into the barrel and shaking the powder into the water. The tip of the piston may then be inserted, acting as a cork, and a little agitation will soon drive the powder into solution. The procedure then consists of entering the vein in the manner described in the directions for injecting arsphenamin, and injecting the solution slowly into the blood stream. In young infants, in whom entry into the usual veins employed, is difficult, recourse may be made to the veins of the scalp.

THE INTRAVENOUS INJECTION OF MEDICAMENTS

The Equipment.—

1. A glass syringe of 1 c.c. or 2 c.c. capacity.
2. A well fitting needle of about 25 gauge.
3. Iodin solution, 2%.
4. Alcohol, 50%.
5. Sterile sponges.

The Site for Injection.—

1. The longitudinal sinus.
2. The external jugular vein.
3. At the bend of the elbow, (median-basilic or median-cephalic veins).

Drugs.—

In emergency, the heart stimulants.

1. Strophanthin, $\frac{1}{1500}$ grain.
2. Adrenalin, (1 to 1000), 3 to 5 minims.
3. Ether, 3 to 5 minims.

Quinine hydrochloride, colloidal silver, bichloride of mercury, the iodids, the salicylates and hexamethylenamin have been recommended for intravenous injection, but their administration by this method is not often necessary during infancy.

The Procedure.—

1. Insure sterility of syringe and of the solution to be injected.
2. Cleanse area with green soap solution, apply iodin and alcohol.

3. Insert needle, attached to the syringe containing the solution, into the vein. (See section on intravenous injection.)

4. Make slight traction on the piston of the syringe. As soon as the vein is entered, blood will flow into the syringe.

5. Inject contents of the syringe slowly.

6. Make slight temporary pressure on wound with a piece of gauze. No dressing is necessary.

INTRAVENOUS INJECTION OF SERUMS

Available Serums.—

1. Antidiphtheritic serum.
2. Antimeningococcic serum.
3. Antidysenteric serum.
4. Antistreptococcic serum.
5. Antipneumococcus serum (Type I).

No serum should be administered intravenously until the patient has been first desensitized by the subcutaneous injection of 1 c.c. or 2 c.c. of the serum to be given. If no anaphylactic symptoms appear, the full dose may be administered intravenously within one hour.

Serums, particularly the antistreptococcic and the antidysenteric, should be well diluted, for the reactions that follow the injection of concentrated solutions are often very severe.

The technic of intravenous injection of serums differs in no manner from that described for the intravenous injection of normal salt solution, (page 483) with the following exception: the serum should be warmed to a temperature not to exceed 102° and added to the container of the normal salt solution as it is flowing into the vein. Just as the last of the diluted serum is disappearing from the container into the tubing, the container should be partly filled with normal salt solution at the proper temperature in order that all the serum may be forced into the vein and the patient be insured the full dose.

INTRAMUSCULAR INJECTION OF MEDICAMENTS

The Equipment.—

1. A glass syringe of 1 c.c. or 2 c.c. capacity.
2. A well fitting needle of 26 or 27 gauge.
3. Iodin solution, 2%.
4. Alcohol, 50%.
5. Sterile sponges.

The Site of Injection.—

1. The triceps muscle (Fig. 4).
2. The gluteus muscle (in infants past the diaper-soiling stage).

Drugs.—

Strophanthin, ($\frac{1}{1500}$ grain); Adrenalin, (1 to 1000, 3 to 5 minims); Camphor (10 per cent) in sterile olive oil, (6 to 10 minims); Caffein Sodium Benzoate, ($\frac{1}{4}$ grain); Quinine Hydrochloride, Mercury, the Iodids, and neoarsphenamin may be so administered.

The Procedure.—

1. Cleanse the selected site and apply iodin and alcohol.
2. Put the skin of the area slightly on the stretch.
3. Plunge the needle deep into the muscle, and inject the solution slowly.
4. Make slight pressure and massage over the area for a few seconds. No dressing is necessary.

INTRAMUSCULAR INJECTION OF WHOLE BLOOD

The administration of this therapeutic agent is simple and easy, and it may be done at the bedside without difficulty. It is not necessary that the bloods of the donor and the recipient be

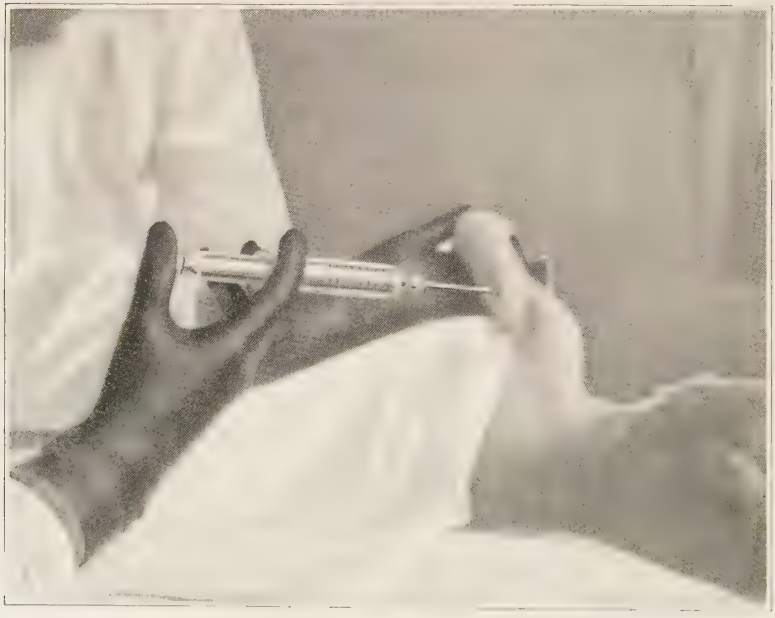


Fig. 4.—Intramuscular injection (triceps).

grouped; the only precaution that should be taken is the insurance that there is no syphilitic or other infection in the donor. Intramuscular injection of whole blood is the method of choice in the treatment of hemorrhagic disease of the newborn. It is also useful in treating malnutrition, secondary anemias, and blood depletion in older children.

Choice of Donor.—

1. Parent of the child.
2. Other persons.

Fig. 5.—Intramuscular injection (gluteus).

The Site of Injection.—

1. The triceps muscle (Fig. 4) (site of choice).
2. The gluteus muscle (in infants who have passed the diaper-soiling stage) (Fig. 5).

The Equipment.—

1. A glass syringe of 20 c.c. to 30 c.c. capacity.
2. Two intravenous needles, size 18, that will fit snugly onto the nozzle of the syringe.

3. A piece of rubber tubing, ⅛ inch in diameter and 18 inches long to be used as a tourniquet.

4. Sterile towels.

5. Gauze dressings.

6. Adhesive plaster.

7. Green soap.

8. Alcohol, 50%.

9. Iodin, 2%.

The Procedure.—

1. Wrap the patient in a sheet restraining the extremities and leave the selected site exposed. Lay the patient on a table.

2. Cleanse the area with green soap solution, dry with alcohol, apply iodin and remove iodin stain with alcohol.

3. Cover the area with a sterile towel.

4. Seat the donor near the patient.

5. Place the tourniquet on the upper third of the donor's arm, tighten just enough to interfere with the venous flow, and leave in dependent position for a few minutes.

6. Cleanse the area at the bend of the elbow with green soap solution, dry with alcohol, apply iodin and remove the iodin stain with alcohol.

7. Attach the needle to the syringe and insert the tip into the donor's vein, preferably pointing the needle toward the hand.

8. Make slight traction on the piston of the syringe. When blood appears in the syringe, increase the traction and with the tourniquet still tightened, withdraw the amount of blood required.

9. Have assistant loosen tourniquet, and make slight pressure with a piece of gauze over the wound and after a few moments apply a dressing.

10. Remove the needle from the syringe and replace it with another which is clean and sterile and plunge the fresh needle deep into the muscle at the site selected.

11. Inject the blood into the body of the muscle with considerable pressure. The whole operation should consume only a very few minutes and the blood does not have time to coagulate.

12. Withdraw the needle and fasten a sterile dressing over the stab wound with a strip of adhesive plaster.

13. Massage the muscle gently for several minutes to aid in diffusion and to hasten absorption of the blood.

SUBCUTANEOUS INJECTION OF FLUIDS

Equipment.—

Syringe Method.—

1. A glass syringe of 20 c.c. to 30 c.c. capacity.
2. A needle, size 19 or 20, 2½ to 3 inches in length. The base of the needle should snugly fit over the nozzle of the syringe.
3. Sterile basin.
4. Thermometer.
5. Green soap.

Fig. 6.—Subcutaneous injection (axilla).

6. Alcohol, 50%.
7. Iodin solution, 2%.
8. Sterile towels and dressings.
9. Adhesive plaster.

Gravity Method.—

1. A needle, size 19 or 20, 2½ to 3 inches in length, preferably of the "slip connector" type.
2. Three feet of ⅛ inch rubber tubing; divide and join again with,
3. Glass connector.
4. Glass container, preferably a Kelly flask.

5. Thermometer.
6. Green soap.
7. Alcohol, 50%.
8. Iodin solution, 2%.
9. Sterile towels and dressings.
10. Adhesive plaster.

The Site.—

1. The axillæ, (preferably).
2. The loose tissues of the abdomen.
3. The buttocks (in children who have passed the diaper-soiling stage).

The Procedure.—

1. Wrap the child in a sheet, restraining the arms and legs and leaving the selected area exposed. If the axillæ are chosen, have assistant hold the arms extended above the head.

2. Cleanse the site with green soap solution, dry with alcohol, apply 2 per cent iodin, and remove the iodin stain with alcohol.

3. Drape the area with sterile towels.

4. If the axillæ are chosen, pick up a fold of skin at about the level of the sixth rib in the midaxillary line, push the needle through the skin at the base of the fold. The point of the needle should be directed toward the apex of the axilla.

5. Inject a little fluid to distend the subcutaneous tissues before carrying the needle upward to its full length. If the needle is run parallel to the midaxillary line, there is no danger of puncturing a blood vessel.

6. Inject 75 c.c. to 125 c.c. into each axilla in this manner by refilling the syringe as often as necessary. Very gentle massage tends to increase diffusion of fluid in the tissues.

7. Withdraw needle, make slight pressure over the stab wound with a piece of gauze for a few moments, and apply sterile dressing fastened into place with a strip of adhesive plaster.

If the abdominal site for injection is chosen, the steps are identical except that a fold of skin is picked up just below the border of the ribs at about the mammary line. The needle is inserted at the base of the fold in a direction pointing toward the umbilicus.

The gravity method is identical with that just described in which the syringe is used, except that the fluid is placed in a container suspended 2 or 3 feet above the level of the site of injection and connected to the needle with rubber tubing.

INTRAPERITONEAL INJECTION

This method of administering fluid to infants is simple, rapid and effective. It is usually painless and it is not productive of shock. With proper technic it is devoid of danger. As a means of combating dehydration in infants, it is invaluable. Either the gravity or syringe method may be used for injecting the fluid.

Fig. 7.—Intraperitoneal injection (syringe method).

The **fluids** available for injection by this method are:

1. Normal salt solution.
2. Glucose solution.
3. Ringer's solution.

The **equipment**, if the syringe method be used, consists of:

1. An intravenous needle, size 16 or 17, with short bevel.
2. A metal connector. This is desirable, but not necessary if the base of the needle fits well over the nozzle of the syringe, and if a flexible connection between the syringe and the needle is not desired.

3. Another metal connector that will fit around the tip of the glass syringe and a piece of rubber tubing 3 inches long and ⅛ inch in diameter to connect the two, and thereby make a flexible connection between syringe and needle.

4. A glass syringe of 30 c.c. to 50 c.c. capacity.

5. A sterile basin to contain the fluid to be injected.

6. A thermometer for testing the temperature of the fluid.

7. Green soap solution.

8. Alcohol, 50%.

9. Iodin solution, 2%.

10. Sterile towels.

11. Sterile dressings.

12. Adhesive plaster.

The **equipment,** if the gravity method is used, consists of:

1. An intravenous needle, size 16 or 17, with short bevel.

2. A metal connector that will fit snugly into the base of the needle and inside the rubber tubing.

3. A piece of rubber tubing, ⅛ inch in diameter and about 3 feet long. The tubing should be cut in two in its lower third, and the two parts joined by a glass connecting tube of the same diameter. This glass connector allows the operator to determine if the fluid is running.

4. A graduated glass container of 500 c.c. or 1000 c.c. capacity. A Kelly flask serves the purpose well.

5. A thermometer for testing the temperature of the fluid. in the container.

6. A medicine glass for testing the temperature of the fluid as it flows out of the tube.

7. Green soap solution.

8. Alcohol, 50%.

9. Iodin solution, 2%.

10. Sterile towels.

11. Sterile dressings.

12. Adhesive plaster.

Site of Injection.—

Abdominal wall, median line, ¾ to 1½ inches below the umbilicus.

The Procedure.—

(a) Empty the bladder.

(b) Enfold the infant's pelvis and legs in a large towel;

pin snugly; restrain the arms across the chest in the same manner. Lay the patient on his back on a table.

(c) Cleanse the abdomen with green soap solution, wash with 50 per cent alcohol, dry thoroughly, paint the lower abdomen with 2 per cent iodin solution, and remove the iodin stain with alcohol.

(d) Drape the abdomen with sterile towels.

(e) Pick up a fold of skin and integument of the abdomen ¾ to 1½ inches below the umbilicus, with thumb and finger of the

Fig. 8.—Intraperitoneal injection (gravity method).

left hand, insert the needle into the fold of skin thus held, and push it in an upward direction through the abdominal wall.

In a thin child, the point of the needle can be felt as it passes the peritoneum, between the thumb and index finger.

As soon as the point of the needle enters the peritoneal cavity, the base of the needle should be depressed in order that the point may not extend into the cavity at a dangerous angle. The fold of skin and integument should be dropped at this time, and no fear need be entertained that an intestine has been injured.

(f) If the operator has an assistant, the syringe may well be

handled by him. It is not necessary that the assistant be provided with rubber gloves so long as he takes pains to keep the tip end of the syringe uncontaminated. The assistant should fill the syringe from the sterile basin containing the injecting fluid, the operator holds the connector and the assistant inserts the tip of the syringe into the connector and injects the fluid. This process is repeated until the desired amount of fluid is injected. Care should be taken that the fluid to be injected is maintained at a temperature of at least 100°. This level can be maintained by setting the sterile basin containing the fluid in a water-bath.

(g) In case the operator has no assistant and desires to use the syringe method, the short rubber tube and metal connectors may be dispensed with, and the syringe connected directly with the needle. The operator then fills the syringe from the sterile basin, himself. No harm will be done in dropping the base of the needle on the abdomen each time disconnection is made in order that the syringe may be refilled.

(h) If it be desired to gravitate the fluid into the peritoneal cavity, the container with the fluid is suspended or held about 2 feet above the level of the site of injection. The fluid is started flowing through the tube and a few c.c. are run into a medicine glass containing a thermometer. If the solution is found to be about body temperature, the connection is made with the needle and the fluid is then allowed to flow into the abdomen. From 100 c.c. to 300 c.c. may be given at one time.

COLLECTING BLOOD FOR A WASSERMANN TEST

The Equipment.—

1. Glass syringe, 2 c.c. capacity, and
2. Needle, size 25 or 26, the base to fit around nozzle of syringe perfectly; or
3. Special collecting tube, devised by Lyon. (Fig. 9.)
4. Lancet, cutting needle, pen point or special puncturing instrument.

The Site.—

1. The longitudinal sinus.
2. The external jugular vein.
3. At the bend of the elbow.
4. Skin surface of the heel or toe.

The Procedure.—

If a vein is selected (site of election).

1. Fit needle on the syringe and prepare area with iodine and alcohol.

2. Apply tourniquet lightly; enter the vein with the point of the needle, make slight traction on the piston of the syringe. As soon as blood appears in the syringe, increase traction and withdraw the required amount, about 5 c.c.

If a stab-wound method is chosen (site of necessity).

1. Cleanse the skin, apply iodin and alcohol. Dry thoroughly.

2. Make generous stab-wound in heel. The flow of blood

Fig. 9.—Capillary tube for collecting blood. (Lyon.)

through the wound will be facilitated by making pressure around the lower third of the leg.

3. As a pool of blood accumulates at the stab-wound, insert the tip of the Lyon tube into the margin of the pool. The blood flows by capillary attraction down the tube and into the bulb, replacing the air which escapes through the air hole situated near the tip.

This collector may be very readily made by taking a piece of ¼ inch glass tubing about 8 to 10 inches long and fusing one end in the flame. As the melted glass accumulates at the end, a small bulb is blown. The other end is drawn out to a capillary tip.

The tube, which is now sealed at both ends, is gently warmed and the flame is applied intensely at a point about 1 inch back from the base of the tip. The hot expanding air of the interior of the tube blows out through the heated glass where the flame is being applied, thus making the air-hole. The end of the capillary tip is then broken off and the tube is ready for use. After the tube has been filled with blood, if it is desired, the air-hole and the tip may be sealed off.

The ordinary Wright's capsule may be used to collect blood if desired; often it has the advantage of expediency.

DETERMINATION OF COAGULATION TIME (RODDA)

1. Cleanse the heel of the infant with green soap solution; sponge with ether and allow to dry.

2. Have in readiness two watch crystals of equal size and a No. 6 lead shot, all cleaned with soap, alcohol and ether.

3. Make a generous stab wound in the infant's heel.

4. Wipe off the first flow and allow blood to drop without any pressure into one of the crystals containing the shot until a small pool has accumulated.

5. Cover with the other crystal and gently roll the shot every 30 seconds until coagulation occurs. (Above 10 minutes is considered pathological.)

LUMBAR PUNCTURE

The Equipment.—

1. The needle. (Fig. 10.) A desirable lumbar puncture needle is 6 to 7 centimeters long, 1 millimeter in caliber, has a short bevel and carries an accurately fitting obturator. It is especially necessary that the bevel of the obturator and that of the needle be identical. The needle should have a heavy base. It should be of steel and only slightly flexible. Its point should be a cutting edge. A corroded or rusty needle should not be used.

2. Two clean, clear, sterile test tubes.

3. Sterile towels and dressings and gloves.

4. Adhesive plaster.

5. Green soap.

6. Iodin solution, 1 or 2%.

7. Alcohol, 50%.

Fig. 10.—Intraspinal needle.

Fig. 11.—Method of holding intraspinal needle.

The Site.—

1. The fourth lumbar interspace, median line (choice). (Fig. 12.)

2. The third lumbar interspace, median line.

Position of Patient.—

To secure proper position and immobility of the child is a very important step of the operation. The assistant grasps with the right hand the back of the patient's neck, close to its shoulders. With the left, she grasps the child's lower thigh just above the knee and flexes it sharply on the abdomen. (Fig. 12.) This

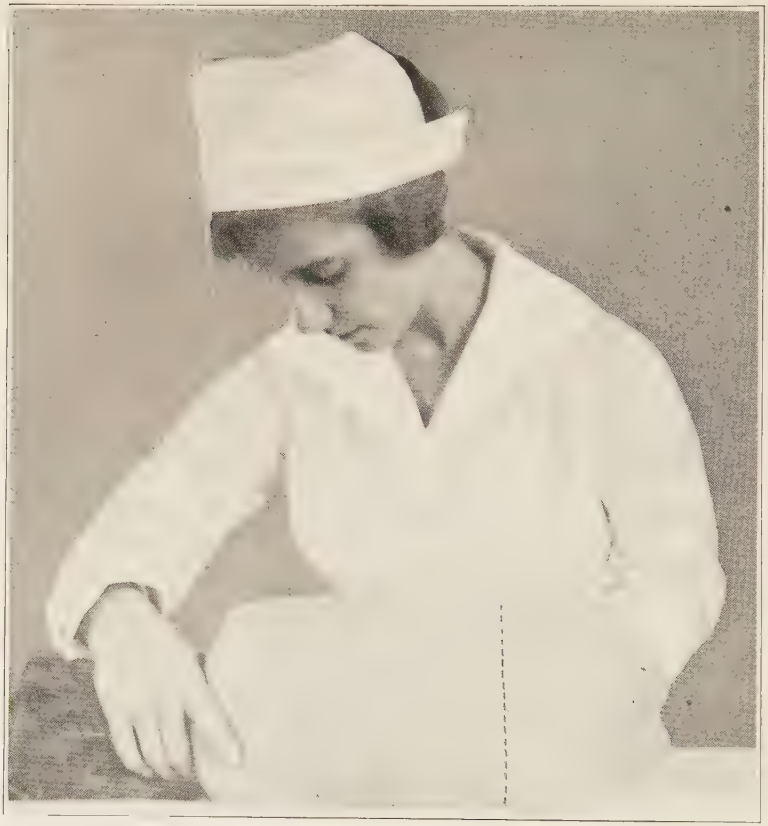


Fig. 12.—Method of holding child.

maneuver will cause the assistant's forearm to hold the uppermost thigh in a like position. Care must be taken that the lower shoulder and the lower hip be kept on the same plane in order to prevent torsion of the spine. If twisting is permitted, the spaces are thrown out of alignment, and it is almost impossible to enter the needle between the bodies of the vertebræ.

The greatest possible degree of flexion of the spine should be attained at the moment the operator is entering the canal. Once the

needle is in position, the assistant may allow some extension of the child's back, particularly where nuchal and vertebral muscular rigidity is a feature. The assistant delays attempting to put the child in the final position until the operator is quite ready to make the puncture; for otherwise, fright and struggling may increase the difficulty of the procedure. The lateral position is always to be used for young patients.

The Procedure.—

1. Cleanse the area with green soap solution, dry with alcohol, apply 2 per cent iodin solution and remove iodin stain with alcohol.

2. Locate the fourth interspace by drawing a line from one iliac crest, across the back to the other. This line may be made with a small cotton swab dipped in iodin solution if desired. (Fig. 12.) This is not essential but it is sometimes helpful.

3. Drape the area with sterile towels.

4. Have the assistant bring the child to the edge of the table and produce the maximum flexion of the spine possible, without undue force.

5. Grasp the needle between the thumb and index and middle fingers of the right hand with the base resting firmly against the palm. (Fig. 11.) The thumb nail of the left hand is used as a guide and the needle is passed through the skin at right angles to the surface. Care must be taken that the right hand holding the needle is kept on the same plane as the top of the table, (assuming that the patient is properly held.) If the hand is raised or depressed, the point of the needle may transfix the thecal space at an angle or miss it altogether.

6. Once the skin is passed, carry the hand holding the needle slightly toward the buttocks so that the needle enters between the spinous processes at an angle of 30 to 35 degrees. With a steady but delicate pressure, force the needle on to a depth of ¾ to 1¼ inches, depending on the age of the child. When it enters the dura, the sudden release of tissue resistance gives an unmistakable feeling.

7. Withdraw the obturator. Fluid should flow at once; if it does not, without entering any further, turn the needle one-half around. This may clear the channel. If it does not, push the needle on a millimeter or two further.

8. If the space has been successfully entered and the cerebrospinal fluid pressure is normal and the child quiescent, the fluid

should flow at the rate of from 20 to 30 drops per minute. Pressure may be so heightened that the fluid will spurt out, or it may flow at the rate of only 3 or 4 drops per minute.

9. Collect the fluid in sterile, clear, glass test tubes. (Fig. 13.)

10. If visible blood is present in any large amount, the results of cytologic and chemical examination will be misleading; but the pressure conditions in the subarachnoid spaces may be judged and the presence of bacteria can be determined both by smear

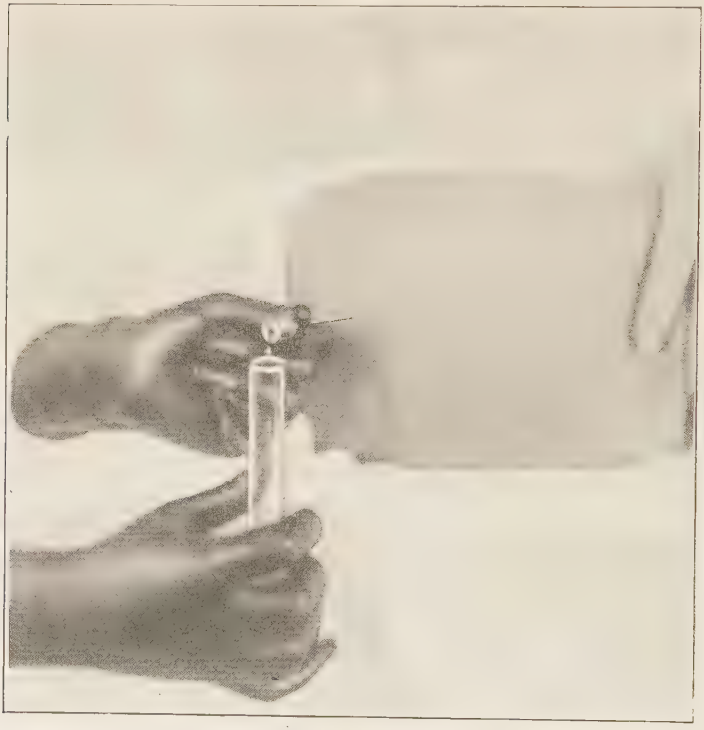


Fig. 13.—Collecting spinal fluid.

and by culture. If the collected fluid be uncontaminated by blood, it should be sent immediately to the laboratory to have a cytologic estimation made and to be examined chemically and bacteriologically. In the absence of blood, a fluid that is not perfectly clear is presumptive evidence of a septic process affecting the meninges.

11. The fluid that appears at the base of the needle may be so thick with pus that it flows with difficulty. In such an instance, gentle pressure with a glass syringe may be made. Often a small

accumulation of pus cells will be removed from the needle and the fluid may then flow more freely. If it be evident that this pressure is insufficient, careful injection of a few c.c. of normal saline solution may facilitate the evacuation of the pus.

12. Withdraw the needle by a quick movement, apply a sterile dressing with an adhesive strap.

INTRASPINAL SERUM INJECTION

Serums Available.—

1. Antimeningococcic serum.
2. Antitetanic serum.

The Equipment.—

1. Lumbar puncture needle.
2. A metal connector accurately fitting into base of needle.
3. Rubber tubing, ⅛ inch in diameter, 1 inch long.
4. Glass connector, ⅛ inch in diameter.
5. Rubber tubing, ⅛ inch in diameter, 3 inches long.
6. Glass container of about 20 c.c. capacity. The barrel of a glass syringe will answer the purpose.

Most biological pharmaceutists provide the above equipment in the package containing the serum. However, the serum can be obtained in bottles.

7. Sterile basin containing sterile water at 104° to 106° to serve as a water-bath for warming the fluid. Hot solutions will coagulate the serum.
8. Thermometer.

The Procedure.—

1. Open the package under aseptic precautions.
2. Place the container in warm sterile water maintained at about 104°.
3. Cleanse area and apply iodine and alcohol. Drape with towels.
4. Have assistant assemble apparatus, exhaust the air from the tube by balancing the tip of the tube against the container.
5. Make lumbar puncture. Allow 30 c.c. to 60 c.c. of fluid to escape. This should be done while the apparatus is being assembled.
6. Connect the metal connector on the end of the tube with the base of the needle and allow the fluid to slowly gravitate into the canal. (Fig. 14.)

7. Should signs of respiratory failure ensue, depress the glass container and allow the fluid to return.

8. If there are no untoward symptoms, withdraw the needle sharply, apply sterile dressings fastened into place with adhesive strips. Return the child to bed and have the foot of the

Fig. 14.—Gravitating serum into the spinal canal.

bed raised on blocks in order to gravitate the fluid toward the cisternæ.

(NOTE: Always inject somewhat less serum than the amount of spinal fluid withdrawn. In cases of meningococcic meningitis and of tetanus, intravenous injections of serum should be made as well as intraspinal.)

PUNCTURE OF CISTERNA MAGNA (AYER)

1. Place the patient in the lateral position as for lumbar puncture.

2. Shave the lower occipital region, wash with green soap solution, pat dry, and apply 2 per cent or 3 per cent iodin solution; remove the iodin with alcohol if desired.

3. Take care to maintain the alignment of the head and the vertebral column in order that there may be no torsion of the spine.

4. Flex the head moderately.

5. Anesthetize the area locally if desired.

6. Select a lumbar puncture needle of about 18 gauge.

7. Place the thumb of the left hand on the spine of the axis.

8. Insert the needle at the midline just above the tip of the thumb.

9. Force the needle upward and forward in the line of the external auditory meatus and the glabella. Aim a little higher than the auditory meatus and when the needle strikes the occiput, depress the point slightly. In a child one year old, the cisterna lies about five-eighths to one inch from the surface. When the dura is pierced, a distinct "give" is felt as in a lumbar puncture.

NOTE: This method of approach to the meninges is very valuable, (1) in the diagnosis of meningitis when adhesions have blocked the passage of fluid into the spinal canal, and (2), as a means of placing a curative serum in direct contact with the basal meninges. The procedure should be attempted only after experience has been gained on the cadaver.

VENTRICULAR PUNCTURE

Purposes of Operation.—

1. To inject antimeningococcic serum.

2. To withdraw cerebrospinal fluid and reduce intraventricular pressure.

3. To inject dyes for diagnostic purposes.

Site of Operation.—

In infants with open fontanels: At the apex of the lateral angle of the anterior fontanel.

In hydrocephalic infants with wide open sutures: In the upper parietal region, just above the motor areas.

In infants with closed fontanels: Through a trephine opening about 2 inches above and 2 inches behind the external auditory meatus.

The Equipment.—

1. A Cushing brain trocar.
2. An intravenous needle of about 1 millimeter bore and 3½ to 4 inches long.
3. An outfit as described on page 511 for gravitating antimeningococcic serum into the spinal canal.
4. A glass syringe.
5. *Neutral* solution of phenolsulphonephthalein containing 6 mg. of the dye to the cubic centimeter. (*Neutral* reaction is imperative.)
6. Safety razor.
7. Sterile towels, dressing bandages, and rubber gloves.
8. Green soap, iodin solution 2 per cent, alcohol, 50 per cent.

The Procedure.—

1. Wrap the child in a sheet, restraining arms to sides and lay it on the table with the head extending just over the edge. In case trephining is necessary, see any standard work on surgery.
2. Shave the scalp over the fontanel.
3. Cleanse the area with green soap solution, dry with alcohol, apply iodin and remove iodin stain with alcohol.
4. Insert the needle through the skin and meninges and at right angles into the brain substance and push it in the direction of the lateral ventricle of the same side. The depth of the ventricle from the surface will depend on the size of the child and the degree of hydrocephalus if it be present.
5. If the puncture is made for the purpose of reducing intracranial pressure, allow the fluid to flow until it ceases running under high pressure. If the puncture is made to inject antimeningococcic serum, withdraw as much fluid as will flow freely, fit the connector after the air has been exhausted from the tube, and allow the warmed serum to gravitate from the container into the ventricle. If the puncture is made for the purpose of injecting dyes for diagnostic purposes, fit the nozzle of a glass syringe containing the dye into the base of the needle and inject the dye. Injection of the full amount of the dye may be insured by withdrawing a little cerebrospinal fluid and reinjecting.
6. Withdraw the needle and apply sterile dressings.

DIAGNOSIS OF TYPES OF HYDROCEPHALUS

Equipment.—

1. Long intravenous needle whose base will fit accurately around the nozzle of a syringe. A spinal puncture needle is satisfactory.
2. Spinal puncture needle.
3. Two test tubes.
4. Glass syringe, 2 c.c.
5. One c.c. ampoule *neutral* phenolsulphonephthalein solution containing 6 mg. of the dye to the cubic centimeter. The ordinary solution of alkaline phenolsulphonephthalein used in kidney function tests is dangerous.
6. Sodium hydrate solution, 25 per cent.
7. Sterile towels, sponges, gloves.
8. Green soap.
9. Alcohol, 50%.
10. Iodin, 2%.
11. Collodion.

The Site of Injection.—

1. Lateral angle of the fontanel, 1 to 2 inches on either side of the median line, for lateral ventricle.
2. Fourth lumbar interspace, median line, for lumbar puncture.

The Procedure.—

1. Wrap the child in a sheet, restraining the arms to the chest, leaving the lumbar region exposed.
2. Lay the patient on his side on a table. The top of the head should extend just over the end of the table.
3. Shave the area over the anterior fontanel.
4. Cleanse with green soap, dry with alcohol, apply 2 per cent iodin and remove iodin stain with alcohol.
5. Cover the area with a sterile towel.
6. Cleanse the lumbar area, dry with alcohol, apply iodin, and remove iodin stain with alcohol.
7. Do a lumbar puncture. (See page 506.)
8. After the fluid appears at the base of the needle, replace the obturator to prevent the fluid from escaping.
9. Have assistant hold the lumbar puncture needle in place while the dye is being injected into the ventricle.
10. Fill the glass syringe with the dye *exactly* to the 1 c.c. mark. Take care that there is no leakage around the piston.

11. Make ventricular puncture. For technic see page 513.

12. After the ventricular fluid appears at the base of the needle, withdraw 1 c.c. of fluid and inject 1 c.c. of dye into the ventricle. Withdraw a syringeful of cerebrospinal fluid and reinject. Repeat this process several times. In this manner, all the dye is washed out of the syringe and into the ventricle.

13. Withdraw the needle from the ventricle and apply collodion dressing.

14. Wait 2 minutes and then withdraw the obturator from the needle in the spinal canal and allow a few drops of fluid to run into a test tube containing 2 c.c. to 3 c.c. of 25 per cent sodium hydroxide solution. When this solution turns pink, it is an indication that some of the dye has passed into the spinal subarachnoid spaces. The normal time for appearance of the dye in the spinal fluid according to Blackfan and Dandy, is 6 to 12 minutes. Naffziger thinks this time limit is too short. At any rate, make observations at 5 minute intervals for 40 minutes. If no dye has appeared by this time, it is safe to assume that the patient has an obstruction between the ventricular system and the spinal subarachnoid spaces.

15. If the dye does appear within a normal time limit, and clinically, the child has hydrocephalus, it can be assumed that there is an interference with the absorptive processes in the subarachnoid spaces and that the type of hydrocephalus is the communicating.

If it be desired to determine the degree of involvement of the absorptive areas in the communicating type of hydrocephalus, it will be necessary to estimate the amount of dye absorbed in a given time by the circulation. This can be done by determining the amount of dye that is excreted in the urine in a fixed period of time. A preliminary kidney function test should be made to determine whether the kidneys are properly functioning. It is well to wait several days to be certain that all the dye used in this test has been eliminated before injecting stain into the ventricles.

The Procedure.—

1. Catheterize the patient; tie the catheter in position by throwing a long narrow adhesive strap around it at a point near the urinary meatus and fasten it to the thighs. Discard the collected urine.

2. Do a lumbar puncture; withdraw 1 c.c. of fluid, and inject

1 c.c. of *neutral* phenolsulphonephthalein solution, (6 mg. to the c.c.), into the spinal canal. Naffziger calls attention to the importance of withdrawing the same amount of fluid that is injected in order that the pressure balance be not disturbed.

3. Withdraw the needle and apply sterile dressing.

4. Collect a 2-hour specimen of urine.

5. Estimate the amount of dye recovered.

Dye Estimation.—

1. Add to the 2-hour specimen of urine, 25 per cent sodium hydrate solution, drop by drop, until the maximum color is attained.

2. Dilute to 500 c.c. with distilled water.

3. Fill the solution cup of the colorimeter with the diluted urine and estimate the percentage. If desired, the less expensive colorimeter composed of standard solutions in ampoules, may be used.

4. Divide the percentage thus indicated by 2. (A dilution of 500 was made).

5. If the color is too high for reading on the colorimeter, dilute the entire solution with 500 c.c. distilled water and then make reading. (As the dilution is now 1 to 1000, no division is necessary).

A recovery of 35 per cent to 60 per cent of the dye injected into the spinal cord within 2 hours is within normal limits. The amount of dye recovered in the 2-hour specimen can be balanced against these normal limits and the degree of damage to the subarachnoid absorbing areas estimated.

GASTRIC LAVAGE AND GAVAGE

Equipment.—

1. Stomach tube. Most babies will take size 14, English. The largest tube that will pass the esophagus should be used. Small tubes are ineffective and they are often irritating. A soft rubber catheter makes an effective stomach tube if the end containing the eyelet is cut off and the cut end rounded on a piece of sandpaper.

2. Glass connector. Should be the same caliber as the tubing.

3. Rubber tubing, ¼ inch in diameter and 2 feet long.

4. Graduated glass cylinder (Fowler). Funnel may be used if desired.

5. Container (1 quart) with sharp-pouring lip.

6. Sodium bicarbonate solution, 2 per cent, 1 quart.
7. Basin to receive stomach contents.
8. Wooden tongue blade.
9. Cork to insert between teeth.

The Procedure.—

1. Wrap patient in a sheet, restraining the arms to the sides. (Fig. 15.)

Fig. 15.—Gastric lavage.

2. Have assistant hold infant in an inclined position.

3. Measure on tube distance between lips and xyphoid cartilage. Indicate with file mark.

4. Open infant's mouth; if necessary use tongue blade; insert cork between jaws.

5. Exhaust the air in the tube and insert into esophagus until the file mark reaches lips.

6. Lower the cylinder to a point below the level of the stomach; this maneuver allows fluid or gas to escape.

7. Pour the irrigating fluid, 2 to 6 ounces according to the age of the patient, into the cylinder. Hold the cylinder 8 to 14 inches above the stomach level.

8. Lower the cylinder below the level of the stomach and siphon off the fluid.

9. Note in the cylinder the amount of fluid recovered, and compare with the amount given.

10. Repeat the process until the fluid returns clear.

Gavage is rarely indicated without a preliminary stomach washing. After the irrigating fluid has returned clear, the *nutrient* solution should be gravitated into the stomach. While the tube is being withdrawn, pinch it off firmly between thumb and forefinger.

BOWEL IRRIGATION

Equipment.—

1. Rectal tube, number 16, English.
2. Glass connector.
3. Rubber tubing, ¼ inch in diameter, 2 feet long.
4. Funnel or container (douche can or rubber bag).
5. Bed-pan, rubber sheeting or Kelly pad.
6. Tube of petrolatum.
7. Irrigating solution, at 104° (normal salt solution or 2 per cent sodium bicarbonate solution).

The Procedure.—

1. Restrain the child by wrapping arms and legs in large towel.

2. Place patient recumbent over bed-pan or in bath tub. Support legs flexed on abdomen. (Fig. 16.)

3. Insert lubricated tube 2 inches and allow water to run. As it flows and distends the rectum, carry the tube up a distance of 6 to 8 inches.

4. Hold the container at a height of 12 to 18 inches and gravitate in from 6 to 8 ounces. Detach the rectal tube from the rubber tubing and allow the fluid to escape from the rectum; or the child may be allowed to expel it.

5. Repeat the process until the quart of fluid has been used.

Fig. 16.—Bowel irrigation.

Sometimes it is desired to use an enema for the purpose of reducing fever. For this purpose, it is desirable to have two rectal tubes; one number 16, English, and the other number 12, English. One tube is used as with a rectal irrigation outfit and is inserted 6 or 8 inches into the rectum and the other is inserted just inside the anal ring to permit the return flow of

the fluid. The solution used should be at a temperature of 65° to 75°. The rate of flow should be slow and from 3 to 6 quarts may be used in the course of an irrigation. Cool enemas are much used for fever reduction but they are not altogether without possibility of danger from too rapid refrigeration and the production of shock.

RECTAL FEEDING

Rectal feeding is of value in infancy chiefly because of the fluid absorbed, although there is probably some absorption of nutrient material, especially of sugars and possibly of the split products of thoroughly peptonized milk. The bowel should be first washed out as directed on page 521.

Nutrient Enemas.—

1. Peptonized milk.
2. Pancreatized cereal decoctions.
3. Sugar solutions, preferably 10% glucose.

The Equipment.—

1. Rectal tube, size 14 English.
2. Glass connector.
3. Rubber tubing, ½ inch, 2 feet long.
4. Funnel or container.
5. Two pitchers.
6. Tube of petrolatum.
7. Normal salt solution.
8. Nutrient enema.

The Procedure.—

1. Wash out the bowel as described on page 521.
2. Wait 20 minutes for bowel irritation to subside.
3. Lay the patient on the side, preferably the right. Elevate hips.
4. Pour nutrient enema into container and allow it to fill the tube.
5. Lubricate tube.
6. Insert tube into rectum 4 or 5 inches.
7. Hold the container 6 or 8 inches above the level of the rectum.
8. Allow 4 to 6 ounces of fluid to flow in slowly.
9. Remove tube gently and hold nates together for a few minutes.

PROCTOCLYSIS

Solutions Available.—

1. Tap water.
2. Normal salt solution.
3. One per cent sodium bicarbonate solution.
4. Ten per cent glucose solution.
5. One per cent sodium bicarbonate and 10 per cent glucose.

If Week's open method is used: (This is the method of choice).

Equipment.—

1. Soft rubber catheter, size 12 French.
2. The glass part of a medicine dropper for a connector.
3. Rubber tubing, ¼ inch, 3 feet long.
4. Funnel of glass or enamel ware.
5. Holder for the funnel. A metal ring attached to the irrigator stand is preferable but a funnel holder may be improvised by making a bandage sling and suspending the funnel from a bed post.
6. Irrigating can with 6 or 8 inches of rubber tubing attached.
7. Screw control clip placed on the rubber tubing just below the container.

If the closed method is used:

Equipment.—

In this method, the equipment is the same as for the open method with the exception that the funnel is replaced by a special glass, bulb connector which carries within it a capillary tip. Through this apparatus, the rate of flow can be observed. It is important that the glass bulb connector should have a small opening at the upper end of the bulb in order that air and bowel gas may escape; otherwise, their pressure may cause pain and the evacuation of the fluid.

The Procedure.—

1. Wash out the bowel with normal salt solution.
2. Wait one-half hour for the bowel irritation to subside.
3. Fill the funnel and tube with the solution at 102°.
4. Pinch off the tube.
5. Lower funnel to a point on a level with or slightly above the pelvis.
6. Lubricate and insert the tube into the rectum 3 or 4 inches.
7. Fasten the catheter into place with long strips of adhesive

plaster encircling the catheter just outside the anus. The free ends should be carried up onto the thighs and fastened.

8. Start the drip. If the funnel is at the proper level, the fluid in the funnel moves up and down with the child's respiration. If it is placed at a higher level than this, the fluid gushes in and irritates the bowel and as a result, it is not well tolerated.

9. The clip on the rubber tubing which is attached to the container, is set so that the fluid escapes from the container into the funnel at a rate rapid enough to maintain the level of the fluid in the funnel.

10. Keep the fluid, entering the rectum, warm by laying a hot water bag in such a way that the tube in passing across the bed from the funnel to the rectum lies against it. There is no need to attempt to keep the fluid in the container warm.

CATHETERIZATION AND BLADDER IRRIGATION

Equipment.—

1. Velvet eye, soft rubber, male catheter, size 7 to 9, French.
2. Glass portion of a small medicine dropper.
3. Rubber tubing, ⅛ inch, 18 inches long.
4. Glass funnel; or graduated cylinder.
5. Glass syringe, 10 c.c. capacity.
6. Sterile basin to collect urine.

Fig. 17.—Catheterization of female infant.

7. Argyrol solution, 20 per cent.
8. Boric acid solution.
9. Sterile towels, cotton, rubber gloves.

The Procedure.—

1. Restrain patient's arm to sides by wrapping sheet around infant.
2. Wash the buttocks and external genitals with green soap solution.
3. Rinse off with boric acid solution.
4. Separate vulvæ and cleanse orifice with cotton sponges soaked in boric acid. If boy patient, wash corona.
5. Remove excessive fluid by pressing gently with squeezed out cotton dab.
6. In the female infant, the urethral orifice will be seen lying in a little pit of mucous membrane just above and behind the upper margin of the vaginal orifice. Grasp catheter with thumb and finger about 1 inch from its distal end and insert. Pass the catheter *almost directly downward* toward the sacrum as the child lies recumbent. (Fig. 17.)
7. Allow the urine to flow out and to collect in a sterile container.
8. To wash out the bladder after the urine is withdrawn, connect the small tip of the glass connector while the fluid is running with the free end of the catheter.
9. Allow the fluid to run in by elevating the funnel 14 to 20 inches above the level of the symphysis pubis. Allow from 2 to 4 ounces of fluid to flow into the bladder.
10. Lower the funnel or cylinder and siphon out the fluid.
11. Repeat the process as many times as is necessary.
12. If it is desired to instill an antiseptic following the last siphonage, inject 1 to 2 drams of a freshly prepared 20 per cent solution of argyrol, or 5 per cent solution mercurochrome.

METHOD OF TREATING THE VAGINA AND CERVIX

The Procedure.—

1. Place the child in the dorsal position.
2. Cleanse the vulva with a soap solution.
3. Lubricate the speculum (Fig. 18) and insert it gently into the vagina.

4. Remove the obturator.

5. Throw a light against the cervix and make inspection.

6. Insert a rubber catheter into the speculum and pass it to the vault of the vagina. Indirect light may be needed for inspection.

7. Withdraw the speculum to a point where the hymen is kept open.

Fig. 18.—Vaginal speculum.

8. Allow the irrigating fluid to flow through the catheter taking care to insure its ready escape.

9. If desired, pass a triangular strip of gauze soaked in 20 per cent argyrol through the speculum and leave it in the vagina.

URINE COLLECTION

Boy Infants.—

Take a piece of adhesive plaster about 3 inches square and a test tube with a shoulder. Cut a hole in the adhesive plaster just smaller than the diameter of the test tube. Insert the closed end of the test tube through the hole from the sticky side and push it through until the shoulder catches on the margin of the hole. Insert the penis into the tube and stick the adhesive against the thighs and pubis. (Fig. 19.)

Fig. 19.—Collecting urine male infant.

Girl Infants.—

A specially devised metal can of appropriate shape (Fig. 20) makes an effective collector. The opening is placed against the vulva, the diaper is pinned on holding the can in position. Failing this, the glass water-container of a bird cage may be used in the same manner as the above mentioned can. It has the disadvantage, however, that it must be held in place with adhesive straps. Failing these special collectors, a rubber glove may be fastened into place by adhesive plaster.

For 24-Hour Specimens.—

A condom or a large rubber finger-cot is passed through a circular hole in a piece of adhesive plaster, 2 by 4 inches in dimensions. The closed end of the finger-cot is slit slightly and the end of a piece of ¼ inch rubber tubing, 2 or 3 feet long, is passed into it to the extent of ¼ inch. A water tight joint is made by winding an adhesive plaster strip about this junction.

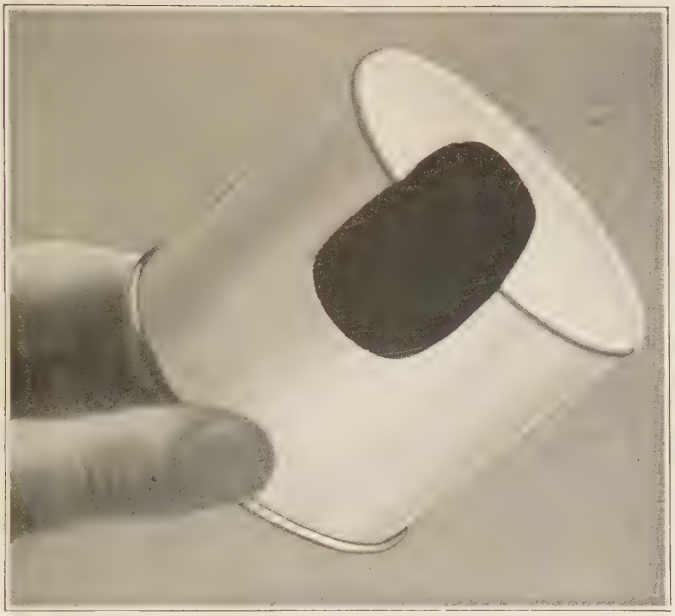


Fig. 20.—Urine collector.

The open end of the finger-cot is now slipped over the penis or around the vulva and made adherent to the pubis, perineum and thighs with adhesive plaster. In the case of a female infant, the adhesive plaster partially closes the vulva and allows the urine to flow down through the finger-cot. The tube is then carried under the bed clothes and outside the bed, the free end is inserted into a nursing bottle which is suspended below the level of the bladder.

IRRIGATION OF THE EAR

Equipment.—

1. Glass portion of a medicine dropper with one-half inch of small rubber tubing attached to the tip.
2. ⅛ inch rubber tubing, 3 feet long.
3. Irrigation can.
4. Kidney basin.
5. Solutions available:
   a. Normal salt solution; or sterile water.
   b. 2% borax solution.
   c. 2% boric acid solution.
   d. 2% sodium bicarbonate solution.

Procedure.—

1. Wrap child in sheet restraining arms to sides. (Fig. 21.)
2. Lay the patient on his back on a table with the head near the edge.
3. Place kidney basin under the ear to be irrigated.

Fig. 21.—Ear irrigation.

4. Fill the container with solution at the desired temperature and allow the fluid to flow. Do not hold the container more than 6 inches above the level of the ear drum.
5. Make upward and backward traction on the pinna of the ear.

6. Insert the rubber-tipped glass tip just within the auditory meatus and allow the fluid to flow gently and escape into the kidney basin.

7. Allow the fluid to flow until the therapeutic purpose is accomplished, whether it be the removal of secretions or the application of heat to the drum.

For ordinary cleansing purposes, the soft rubber, bulb "ear and ulcer syringe" is often amply sufficient. When the ear drum has been ruptured, great care must be taken that the irrigating stream does not force fluid and secretions back into the middle ear.

EAR EXAMINATION AND PARACENTESIS TYMPANI

Equipment.—

1. Ear speculums of assorted sizes.
2. Head mirror.
3. A good light for reflection. The head mirror and light may be replaced with advantage by an electric-lighted otoscope carrying a dry cell in the handle (ample for examination only).
4. Sterile cotton applicators.
5. Von Grafe cataract knife.
6. Aural rubber bulb or glass syringe.
7. Solution of normal salt.

The Procedure.—

1. Wrap the child in a sheet, restraining the arms to the sides. (Fig. 22.)

2. Have an assistant hold the infant on her lap with the ear to be examined placed forward. With one arm and hand the assistant should encircle the patient's pelvis; and with the other hand she should support the head firmly against her shoulder.

3. Place sterilized instruments on a table within reach.

4. Be seated in a chair of sufficient height to bring the reflector of the head mirror on a level with the patient's ear.

5. Place the light in such a position that the reflection from the head mirror is easily and comfortably thrown into the auditory meatus.

6. Pull the ear forward and upward and insert the speculum into the auditory canal.

7. Remove any secretions from the canal with a cotton applicator. It may be necessary to soften the secretions with hydrogen peroxide and wash them out with boric acid solution.

8. Inspect the tympanum. Observe the color, character of the light reflection, visibility and relationship of ossicle attachment, and whether or not there is a bulging of the drum or any part of the walls of the auditory canal.

Fig. 22.—Ear examination.

9. If the drum is bulging, wash out the canal with 2 per cent boric acid solution, wipe out with an applicator dipped in alcohol, dry with a sterile cotton applicator. Insert the tip of the von Grafe cataract knife through the lowest part of the greatest

bulging and move it backward and upward at an angle of about 30° and with the same motion withdraw it. It is doubtful whether local anesthetics applied to the drum are effective. The operation is so brief and the pain, while intense, is of such short duration, that the use of a general anesthetic is not often necessary.

10. Place a cotton plug into the external auditory canal. This plug should be changed frequently in order that the free exit of discharges may not be interfered with.

11. The incision in the drum should be kept open for several days. In order to do this, the ear should be irrigated once or twice a day according to the technic described on page 527.

(NOTE: The occasion for drum puncture is so frequent in pediatric practice that every physician who deals with children should familiarize himself with the procedure. His hand-bag should always contain the necessary apparatus. While it is well to call an aurist when possible, circumstances do not always permit it.)

THROAT EXAMINATION

Equipment.—

1. Tongue depressor, wooden blade or tablespoon.
2. Light.
   a. Daylight.
   b. Electric flash-light.
   c. Head mirror and lamp.

Procedure.—

1. Wrap the child in a sheet restraining its arms to its sides.

2. Have an assistant hold the child erect in her lap, with the child's back to her chest, her right arm encircling its body and her left hand on its forehead, firmly holding the head against her shoulder. If the patient is very ill, he may be examined in bed, but the arms should be restrained in the same manner. The time spent in properly restraining the infant is more than compensated for by the satisfactory examination.

2. Pass the tongue depressor into position. If the child resists by clinching its teeth, make slight pressure with the end of the blade between the lips and the teeth. If this pressure is maintained for a few seconds, the child will part the teeth. No force

is necessary to accomplish this. The blade of the tongue depressor should be passed quickly backward until it passes the faucial pillars. The result will be to make the child gag and at this moment, pressure downward and forward on the base of the tongue will reveal the fauces and pharynx and sometimes even the epiglottis.

3. Direct the light into the throat and inspect tonsils, pillars, uvula, palate, palatal edge, and the posterior pharyngeal wall. Note the swelling, ulceration, exudate and altered relationship.

CULTURE TAKING

Equipment.—

1. Tongue depressor.
2. Tubes of culture medium.
   a. Glucose-agar.
   b. Loeffler's blood-serum.
3. Sterile swabs.
4. Light.
   a. Daylight.
   b. Flash-light.
   c. Head mirror and lamp.

The Procedure.—

1. Wrap the child in a sheet restraining the arms to the sides.

2. Have an assistant hold the patient on her lap with the child's back to her chest and the head held firmly with her left hand across its forehead. (Fig. 23.) The child should face a window if natural light is used.

3. Hold the culture tube between the middle and ring fingers of the left hand with the cotton plug pointing upward.

4. Take the tongue depressor in the same hand and depress the child's tongue. A little continued pressure against the teeth for a few seconds will make the patient open his mouth. Pass the depressor back toward the fauces and when the patient gags, make downward and forward pressure on the blade. The posterior pharyngeal wall and the fauces will then come plainly into view.

5. With the sterile swab in the right hand, touch the tonsils and the faucial ring and if a laryngeal infection is suspected, touch the posterior wall of the pharynx and the epiglottis if possible.

Fig. 23.—Culture taking.

6. Drop the depressor on a paper and with the hand holding the swab remove the cotton plug, taking care not to touch that surface of the plug which goes into the test tube against any object.

7. Gently rub the cotton swab over the slanted surface of the culture medium, taking care not to injure the smooth surface.

8. Replace the cotton plug into the test tube.

9. Burn the swab and boil or burn the tongue depressor.

THROAT IRRIGATION

Equipment.—

1. Rubber catheter, size 12, English. Cut off the tip end.
2. Glass connector.
3. Rubber tubing, ¼ inch, 3 feet long.

4. Irrigation can.
5. Large waste basin.
6. Solution. Choice of:
   a. Normal salt.
   b. Sodium bicarbonate, 2 per cent.
   c. Borax, 2 per cent.
   d. Boric acid, 2 per cent.

The Procedure.—

1. Wrap the child in a sheet restraining the arms to the sides.
2. Lay infant on its side on a table. (Fig. 24.)

Fig. 24.—Faucial irrigation, lateral position.

3. Bring the patient's head over the edge of the table, flex slightly and rotate the face toward the floor.

4. Open the mouth and keep it open with a gag. A wooden tongue blade held on edge between the jaws is sufficient to accomplish this purpose.

5. Pass the catheter close to the lower angle of the mouth and direct it across the floor of the mouth toward the uppermost tonsil until the tip of the catheter is close to the upper pillar.

6. Elevate the container 12 or 14 inches above the level of the

child's head and allow the fluid to flow. If the head is in proper position and the pressure not extreme, the fluid should flow over the upper tonsil and the superior part of Waldeyer's ring, down over the lower tonsil and out through the lower corner

Fig. 25.—Faucial irrigation. Upright position.

of the mouth without entering the pharynx or causing any sensation of choking or distress. Infants and children with painful fauces soon come to appreciate this procedure. It is especially valuable in the anginal forms of scarlatina and in streptococcic sore throats with ulceration. The irrigation may be ac-

complished if desired with the child in the upright position. (Fig. 25.)

To Irrigate Throat While Child Is in Erect Position

The Procedure.—

1. Wrap the child in a sheet restraining the arms to the sides.
2. Hold the infant on the lap with its back to the operator and the head and body held in an *extreme forward flexion.* This position allows the ready escape of the irrigating fluid.
3. Insert the tube into the mouth while the water is flowing.
4. Hold the container about 2 feet above the level of the mouth and allow the fluid to flow. If the child gags and chokes, remove the tube and repeat the process. If the child clinches its teeth, make a steady pressure against the lip for a few seconds; he will then open his mouth.
5. Allow the contaminated fluid to escape from the mouth into a large basin held under his chin.

NASAL IRRIGATION

Equipment.—

1. Soft rubber bulb, "ear and ulcer syringe."
2. Large basin.
3. Solution. Choice of:
   a. Normal salt.
   b. Borax, 2 per cent.
   c. Boric acid, 1 per cent.

The Procedure.—

1. Wrap the child in a sheet restraining the arms to the sides.
2. Seat the child on your lap with its back in the crook of your arm. (Fig. 26.)
3. *Lean well forward* and flex the child's head over the waste basin.
4. With the palm and three fingers of the left hand on the patient's forehead, support the head firmly. Raise the tip of the nose with the free little finger of the same hand.
5. With the other hand, fill the syringe and place the nozzle at the opening of the nostril in a line parallel with the roof of the mouth.
6. Squeeze the bulb gently and make the fluid flow into the

Fig. 26.—Nasal irrigation.

nostril. It should escape through the mouth and the opposite nostril. Repeat the process on the other nostril. If the child is held in the extreme inclined position with head flexed and if only gentle pressure is made on the syringe, there is no danger of fluid running into the Eustachian tube.

EXAMINATION OF CONJUNCTIVA

Equipment.—

1. Lid retractor.

2. Small rounded instrument for everting upper lid. Cotton on applicator will suffice.

3. Light.
   a. Daylight.
   b. Flash-light.
   c. Head mirror and lamp.

The Procedure.—

1. Wrap child in a sheet restraining the arms to the sides.
2. Wipe off secretions with sterile sponge. Moisten with boric acid solution if necessary.
3. With thumb and finger of the left hand, grasp the cilia close to their insertion into the lid, make gentle traction at first downward and then away from the orbit.
4. Place the applicator, held in the right hand, against the outer surface of the upper lid and turn the lid up over it, at the same time removing the applicator. If there is not too much swelling, these two maneuvers will evert the lid. The lower lid is everted in the same manner but with less difficulty.

When the conjunctivæ are greatly swollen, it will be necessary to use *lid retractors.*

The Procedure.—

1. Slip the upper blade of the retractor under the upper lid.
2. Insert the lower blade under the lower lid.
3. Spread the blades gently by turning the screw of the instrument.
4. Carefully avoid touching the cornea because it is readily injured when there is swelling of the conjunctiva.
5. Observe the degree of swelling, abrasions or ulcers on the conjunctiva, the character of the epithelium of the cornea, whether glistening or sodden. Throw the light laterally on the cornea and notice if there is any break in its continuity. Note the pupillary reflex and look for opacities of the lens. Look for color changes about uveal body and changes in character, number, and contour of blood vessels at the uveal margin.

EYE IRRIGATION

Equipment.—

1. Glass portion of a medicine dropper with a small piece of rubber tubing attached to the tip. (Fig. 50, page 574.)
2. Rubber tubing ⅛ inch in diameter and 3 feet long.
3. Irrigating can.
4. Kidney basin.
5. Solution. Choice of:
   a. Boric acid, 1 per cent.
   b. Borax, 2 per cent.
   c. Normal salt.

The Procedure.—

1. Wrap the child in a sheet restraining arms to its sides.

2. Lay the child on the table with the eye to be irrigated lower. (Fig. 27.)

3. Place the kidney basin under the eye.

4. Cover the uppermost eye with a sponge; especially necessary if the discharge be profuse.

Fig. 27.—Eye irrigation.

5. Place the container 10 or 12 inches above the level of the eye and flow the solution across the eye from inner canthus to outer canthus and into the kidney basin. If there is great swelling, it may be necessary to widen the palpebral fissure with a lid retractor.

6. If the other eye is to be irrigated, turn the infant over and repeat the process.

Note: This method of irrigation of the eye is of particular value in the gonorrheal ophthalmias. The restraining sheet guarantees immobility and prevents contamination of the patient's hands.

EYEGROUND EXAMINATION

Equipment.—

1. Ophthalmoscope. A good electrically lighted instrument carrying its own dry cell battery is convenient.

2. Light. A ground glass, high candle-power electric lamp is preferable.

3. Homatropin solution, ½ of 1 per cent.

The Procedure.—

1. Instill a few drops of the homatropin solution into each eye.

2. Wait at least 30 minutes to make the examination.

3. Place the child in position. This may be erect on the nurse's lap or recumbent in bed (Fig. 28) or in the lateral position recommended by Batten. In the Batten position, the child is laid

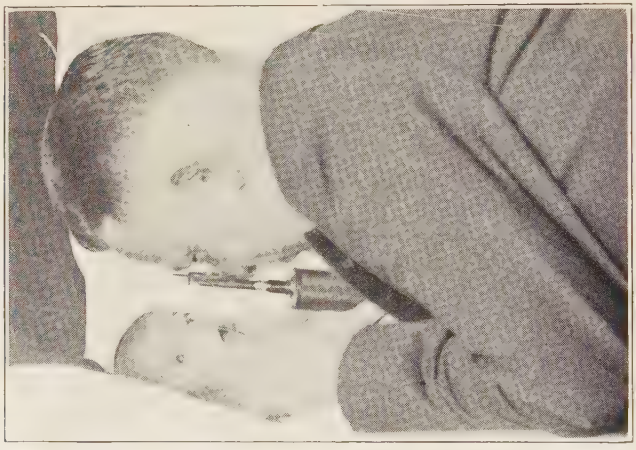


Fig. 28.—Ophthalmoscopic examination.

on its side on a table or bed and held there by a nurse or propped by pillows.

No attempt should be made to go on with the examination until the child is quiet and reconciled to the position. The advantage of this method is that lateral movements of the eye are less frequent and extensive in this position than in the erect or recumbent. The upper eye is the one to be examined. When this is accomplished, reverse the child head for foot, so that the other eye becomes uppermost. For infants under 6 months of age, this is the most satisfactory method.

4. Wait until the child is reconciled to its position. Restraining may increase the child's struggling and the examination may be more satisfactory without wrapping the patient in a sheet.

5. With the ophthalmoscope held in position at the operator's eye, get the red fundal reflex of the child's eye. If the right eye is being examined, the operator should use his own right eye; if the left, his own left.

6. Relax the accommodation of the observer's eye by attempting to focus on an imaginary point about 20 feet directly behind the center of the patient's pupil. Do not attempt to follow the lateral movements of the child's eye. If the patient cries, exercise patience and wait.

7. Observe the appearance of the fundus, the vessels, the macula and the disc as these structures move across the examiner's field of vision and take note of any opacities of the anterior ocular apparatus which interfere with a complete view of the background.

TEMPERATURE TAKING

It is useless to try to take a child's temperature by mouth, and to use the axilla or groin is very inaccurate. Rectal temperature only is accurate.

If the infant is very ill.—

1. Turn the child on its side with back to the edge of the bed.

2. Flex the legs upon the abdomen and hold the child in this position if it tends to struggle.

3. Make slight backward and upward traction on the skin at the posterior margin of the anus. The furrow thus produced leads directly into the anus.

4. Insert the lubricated bulb of the thermometer into this furrow and thence into the rectum.

5. Leave the thermometer in position 2 minutes.

6. Remove, wipe off the lubricant if necessary, and read.

If the child is not very ill.—

1. Remove clothing from buttocks (Fig. 29).

2. Lay the child on its abdomen across the lap.

3. Hold the legs in a semiflexed position if the child struggles. Ordinarily he will remain quiet in this position.

4. With the other hand, insert the thermometer. The best type of rectal thermometer has a bulb shaped mercury chamber.

Fig. 29.—Rectal temperature taking.

SPONGING TO REDUCE FEVER

The Procedure.—

1. Lay rubber sheeting on bed to protect bedding.
2. Place large bath towel over rubber sheeting.
3. Remove infant's clothing; lay patient on the towel. Cover with blanket.
4. Begin sponging with water at about 90°, one part at a time, keeping the parts not being sponged covered with the blanket.
5. Gradually reduce the temperature of the water by the addi-

tion of cold water or small pieces of ice. Reduce not lower than 65°.

6. Sponge for 15 or 20 minutes unless the child's condition becomes unsatisfactory.

7. Pat dry and give alcohol rub if desired.

MUSTARD BATH

Equipment.—

1. Tub.
2. Can of ground mustard.
3. Small cotton blanket or bath towel.
4. Dry bath towels.
5. Hot water bag.

The Procedure.—

1. Make a paste composed of 4 to 6 level tablespoonfuls of ground mustard and cold water.

2. Put enough water at 100° to 105° into the tub to cover the child.

3. Stir the mustard paste into the water.

4. Put child into the tub and cover with a blanket. Support the patient's head outside the blanket.

5. Keep child in bath 5 to 10 minutes.

6. Rinse off by douching patient with pitcher of warm water as he is being removed from the tub.

7. Dry with a warmed towel.

8. Wrap patient in a warmed blanket and put to bed. Pack hot water bottles around him. Leave in blanket for ¾ hour to 1 hour, at which time the infant should be freely perspiring.

9. Take child out of blanket, dry with warmed towel, rub with alcohol and return to bed between sheets.

MUSTARD PACK

Equipment.—

1. A small cotton blanket or large bath towel.
2. Woolen blanket.
3. Rubber sheet.
4. A small tub.
5. Can of ground mustard.
6. Ice cap.
7. Hot water bag.
8. Thermometer.

The Procedure.—

1. Take 7 tablespoonfuls ground mustard and make it into a paste with cold water.

2. Stir this into 5 or 6 quarts of water at 120°.

3. Spread rubber sheet on the table. (Fig. 30.)

4. Spread a dry woolen blanket on top of the rubber sheet, folding the sides over.

5. Have assistant fold the dry, cotton blanket lengthwise into accordion-like pleats, bring the ends together; put the folded blanket into the hot mustard water, insert a stick in the loop and wring the blanket as dry as possible.

Fig. 30.—Mustard pack showing blanket, rubber sheeting, and ice cap in position.

6. Open up the folds of the dry, woolen blanket and have the assistant place the hot cotton blanket inside it in such a manner that it may be quickly unfolded to receive the infant. Cover over immediately with the woolen blanket in order that the heat may be conserved. (While 120° may seem a high temperature, it should be remembered that a great deal of heat is radiated during the wringing-out process, and by the time the child reaches the pack, the temperature will have been reduced to below 110°.)

7. Slip child into pack.

8. Tuck blankets under arms and between the legs. Enfold with rubber sheet.

9. Leave child in the pack for 20 to 30 minutes.

10. Take patient out of pack and rinse it off with plain warm water.

11. While doing this, have an assistant wring a sheet out of plain water at 110° and arrange it in the pack in place of the mustard-saturated blanket.

12. Return the child to the pack.

13. Leave patient in pack 1 hour. Put ice cap to head.

14. Remove infant from pack and pat dry.

15. Return to bed with hot water bottle. If the pack has been successful, the patient should be very flushed and perspiring.

WET PACK

Equipment.—

1. Woolen blanket.
2. Cotton sheet.
3. Rubber sheet.
4. Small tub or large pan.

The Procedure.—

1. Spread the rubber sheet on the table or bed.

2. Place the woolen blanket over the rubber sheet.

3. Wring the bath towel or cotton blanket out of water at a temperature corresponding to the type of pack desired. A cold pack, 70°; a tepid pack, 90°; a hot pack, 110° to 120°. Place the cotton blanket inside the woolen blanket.

4. Elevate the arms above the head and lay the patient on the wet sheet at the junction of the middle and the right ⅓ of the sheet.

5. Draw the right ⅓ of the sheet across the body from right to left. Tuck the upper portion along the right side of the trunk and the lower portion between the legs.

6. Place the arms at the side of the body. Draw the left ⅓ of the sheet over from left to right to envelop arms and entire body. Tuck free edge along the left side of the body and with the lower end enclose the feet snugly.

7. Fold the woolen blanket over from right to left, tucking the right edge along the right side of the body and carrying the left edge over to the left side, tucking under and firmly securing. It is important that the blankets be snugly tucked and so arranged that air is excluded from the patient in the wet pack.

8. Apply a cold wet towel to the head.

9. Leave the patient in the tepid pack for 20 to 30 minutes; in the cold pack 20 minutes; and in the hot pack 30 to 60 minutes.

10. Remove the patient from the pack, sponge with cool or tepid water and rub briskly.

CHEST PACK

Equipment.—

1. Small bath towel.
2. One yard of oil silk.
3. Cotton binder, 8 inches wide and 2 to 2½ feet long.

The Procedure.—

1. Lay the binder flat on the table.
2. Lay oil silk on binder leaving a few inches of the binder free at each end.
3. Wring towel out of cold or tepid water or hot mustard water, according to the results desired.
4. Lay the child on the towel.
5. Elevate the arms and draw the right side of the towel over across the chest and tuck it down along the axillary border.
6. Draw the opposite side over and tuck in along the opposite axillary border.
7. Cover with the oil silk.
8. Bring the binder across and pin loosely.
9. Leave the chest pack in position 30 minutes to 1 hour.
10. Remove pack, sponge with water and give an alcohol rub.

HOT AIR BATH

Equipment.—

1. Two straight back chairs.
2. Two wooden slats, 2½ feet long.
3. One wooden slat 3 feet long.
4. Heavy blankets.
5. Heater—either electric, gas or coal-oil stove not over 2 feet high.

To Assemble.—

1. Place the chairs about 2 feet apart with their backs about 2½ feet from the foot of the infant's bed.
2. Tie one of the 2½ foot slats between each of the chair tops and the foot of the patient's bed.

3. Connect the chair tops with the 3 foot slat. A frame to support the blankets is thus made.

4. Spread a heavy blanket over the frame and allow it to hang over the sides to within a few inches of the floor.

5. Place a pillow on either side of the patient lying in bed.

6. Spread the other end of the blanket over the patient. The pillows should be thick enough to prevent the blanket from touching the patient's body.

7. Tuck the upper margin of the blanket about the patient's neck, thus leaving the child's head outside the enclosed space.

8. Place the heater on the floor at the foot of the bed and between the two chairs. As the surrounding air is heated, it ascends into the enclosed space around the child.

(A metal hood designed to stand on the floor at the foot of the bed and carry the heated air up under the bed clothing can be made by a tin-smith.)

The Procedure.—

1. Spread a rubber sheet on the bed to protect the mattress.

2. Spread a bath towel or folded sheet over the rubber sheet.

3. Wrap the patient in a dry, warm sheet and lay him on the towel. Place pillows alongside.

4. Cover the child with the woolen blanket, carefully tucking it in about the neck.

5. Light the stove and test the temperature in the enclosed space from time to time by inserting the hand under the cover.

6. After the patient begins perspiring, leave him in the bath from 15 minutes to 1 hour according to the indications.

7. Remove patient, pat dry and give alcohol rub if desired.

WATER EVAPORATION OUTFIT

Valuable in Hot Dry Climates to Keep Fever Patients Cool

To Construct.—

1. Make a gabled frame of wood, resembling a roof, large enough to cover the infant's crib. The roof should be about 3 feet above the surface of the bed.

2. Cover the roof-frame with muslin or canvas.

3. Make another roof-frame of similar size, cover it with muslin or canvas and fasten it over the first roof, leaving a space of 6 or 8 inches between the two.

4. Take a piece of small gas pipe of length equal to the ridge of the roof, close one end and bore a few tiny holes throughout its length. Fasten it on top of the upper roof, running alongside the ridge.

5. Connect the open end with a water reservoir (a five gallon tin can will answer the purpose), or with a water tap if one be available.

6. Allow the water to drip over the upper roof, fast enough to keep the canvas constantly wet. The evaporation of the water cools the air in the space between the upper and the lower roofs. The cool air, being heavier than the surrounding hot air, flows downward and around the patient. The use of this improvised apparatus may prove a life-saving measure in treating children with high fevers who live in communities where electric fans and ice are not available.

HOLDING AN INFANT FOR AUSCULTATION AND PERCUSSION

The Method.—

1. Remove all clothing except diaper and stockings.

2. Have mother or nurse hold the child in the erect position with the child's face over her shoulder. (Figs. 31 and 32.)

3. The mother's right hand should be on the infant's buttocks and the left hand on the shoulders and out of the examiner's way. In dealing with contagious diseases, a towel should be draped between the mother's and the infant's faces.

4. For percussion, (Fig. 32), the infant's body should be held away from the mother's as close approximation somewhat changes the percussion note.

CHEST PUNCTURE

Equipment.—

1. Glass syringe of 20 to 30 c.c. capacity.

2. A 3 inch or 4 inch intravenous needle, size 17 or 18, with short bevel. This needle should accurately fit onto the syringe.

3. Pus basin.

4. Sterile towels and dressings.

5. Adhesive plaster.

6. Green soap.

7. Alcohol, 50 per cent.

Fig. 31.—Holding infant for auscultation.

8. Iodin solution, 10 per cent.
9. Iodin solution, 2 per cent.

The Procedure.—

1. Wrap a sheet around the child from the waist downward to restrain the legs.

2. Have assistant hold the infant in an erect position with her right hand holding the buttocks and legs, the left encircling the shoulders and restraining the arms in such a manner that the

Fig. 32.—Holding infant for percussion.

patient cannot interfere with the operator. The arms should be elevated. (Fig. 33.)

3. If it be suspected that the fluid is free in the pleural cavity, locate the seventh interspace in the anterior scapular line and mark the point with 10 per cent iodin solution. If a radiogram indicates the fluid to be localized, mark the nearest available interspace.

4. Cleanse the area with green soap solution, dry with alcohol,

and apply 2 per cent iodin solution. Remove the iodin stain with alcohol except at the point indicated with the 10 per cent iodin.

5. Fit the needle to the syringe and insert the point at the interspace chosen.

6. In order to protect the subcostal artery locate the lower border of the seventh rib with the thumb nail of the left hand and insert the needle pointing slightly downward just below this

Fig. 33.—Paracentesis thoracis.

point. Ordinarily in a thin infant, ¼ to ½ inch penetration will reach the pleural cavity. The presence of an organized exudate or old adhesion may make it necessary to go deeper.

7. Gently withdraw the piston of the syringe to determine if there is fluid in the cavity.

8. Withdraw fluid, if any be found, according to the age and condition of the patient. Place a few cubic centimeters in a sterile test tube for culture. Ordinarily, it is not safe to withdraw more than 100 c.c. at one time.

9. Withdraw the needle with a quick pull and apply sterile dressing, fastening it into place with adhesive straps.

(NOTE: In young children rib resections are contraindicated as the soft and comparatively nonresilient lung tissue has a tendency to collapse. Empyema is best treated by repeated aspirations, checked by fluoroscopic observations.)

THE CROUP KETTLE

Equipment.—

1. Specially devised kettle. (Fig. 34.)
2. Holt croup kettle, composed of an alcohol lamp and a water container with a spout.
3. An electric vaporizer.
4. A tea kettle. (Fig. 35.)

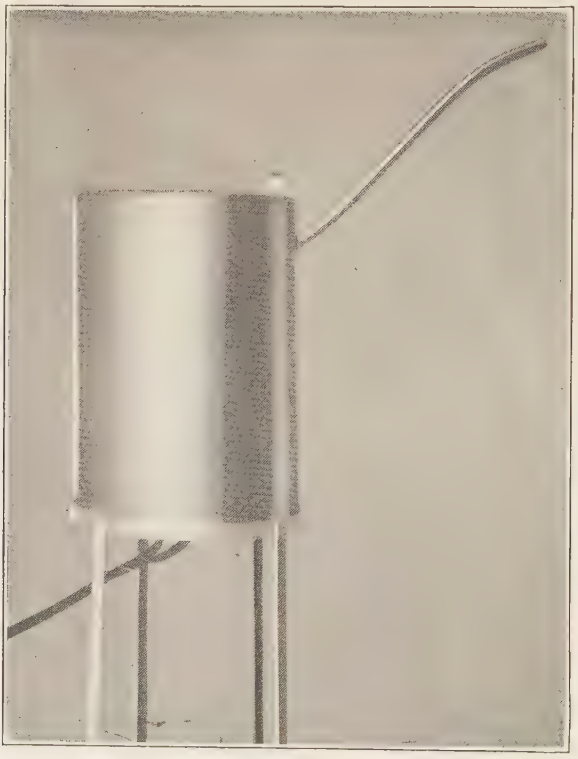


Fig. 34.—Croup kettle.

The Procedure.—

1. Make a tent over the infant's bed by draping a sheet over the crib. An open umbrella, tied to a bed post, makes a good

support. (Fig. 35.) It is important that one or two sides of the tent be left open to insure ample ventilation for the child.

2. Have the kettle boiling and direct the steam under the tent, taking care that the column of steam does not strike the baby's face, and that condensed steam does not drop off the spout onto the infant. A hot flatiron will serve to keep the water in the kettle boiling for a time.

Fig. 35.—Improvised croup kettle and tent.

3. Allow the infant to inhale the steam-laden atmosphere for ½ to ¾ hours.

4. Discontinue the process and repeat if necessary after a few hours. Continuous steam inhalation is to be condemned.

(NOTE: The volatile oils such as oil of pine or eucalyptus or the compound tincture of benzoin may be added to the boiling water if desired, although it is probable that they have little therapeutic effect.)

OXYGEN ADMINISTRATION

Equipment.—

1. Tank containing oxygen. (Fig. 36.)
2. Key to release valve. (Should be attached to the outfit.)
3. Rubber tubing, ¼ inch in diameter, 18 inches long.

Fig. 36.—Oxygen apparatus.

4. Wolff bottle with 2 rubber corks.
5. One-fourth inch glass tubing, 3 inches long.
6. One-fourth inch glass tubing, 6 inches long.
7. Rubber tubing, ¼ inch in diameter, 3 feet long.
8. Funnel, 3 inch.

To Assemble.—

Attach to the outlet of the tank the short piece of rubber tubing. Into the other end of the tubing insert the long glass tubing. Extend the glass tubing through the opening in one rubber cork to within a short distance of the bottom of the Wolff bottle. Fill the bottle one-half or two-thirds full of warm water. Insert the short glass tube just through the opening of the other rubber cork. Attach the long piece of rubber tubing to the free end of the glass tubing. Connect the funnel with the free end of the rubber tube. See that all connections are air tight. The Wolff bottle should be suspended to the tank by a suitable appliance. It is very convenient to have the tank mounted on a specially constructed wheeled-platform with a metal upright support for its attachment. (Fig. 36.)

To Administer.—

Turn the valve and allow the gas to bubble through the water in the Wolff bottle. Count the rate at which the bubbles break at the surface of the water. (Thirty to 50 bubbles per minute.) Administer but 10 to 20 minutes out of every hour for 3 hour period.

The advantages of this method are:

a. The ability to check the rate of flow.
b. The administration of warm and humid gas.
c. Avoidance of irritation of mucous membrane.
d. Economy.

ARTIFICIAL RESPIRATION

The method of Sylvester is the safest for use in infants. Schultze's method is very efficient but it is dangerous in any but experienced hands. Fractured ribs, injury to the spine or ruptured viscera may result from its application.

Sylvester's Method

The Procedure.—

1. Clear the mouth and throat of any secretions or obstructions that may be present. (Fig. 39.)
2. Place the child on its back on the table.
3. Make traction on the tongue if necessary.
4. Extend the arms above the head and hold them in this posi-

tion for a brief space of time giving the lungs opportunity to fill. (Fig. 37.)

5. Bring the arms to the chest cage and exert gentle pressure to force the air out of the lungs. (Fig. 38.) Remember that

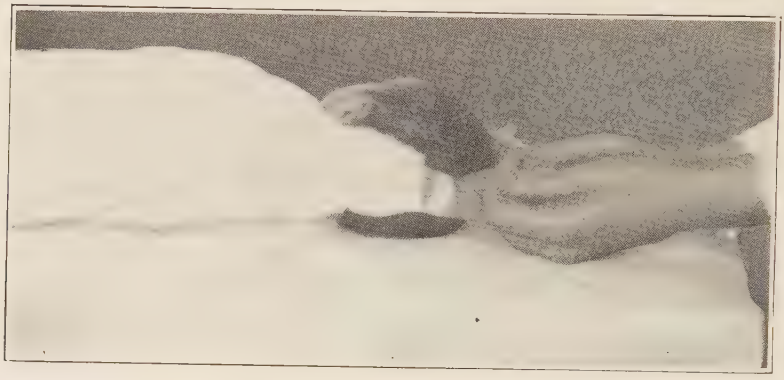


Fig. 37.—Artificial respiration, first step.

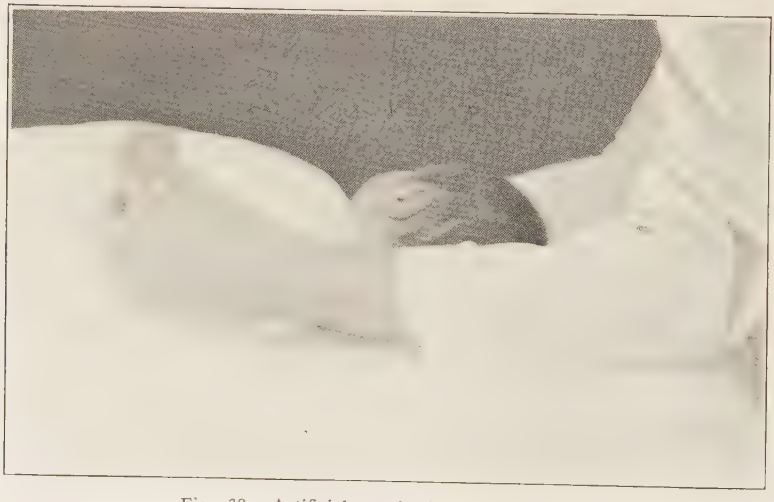


Fig. 38.—Artificial respiration, second step.

a young child's tissues are soft and that too much force may be very easily exerted, especially through stress of excitement.

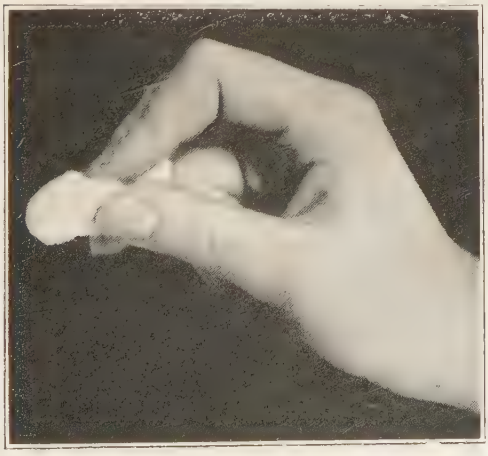
6. Repeat these two maneuvers at the rate of about 20 per minute, that is, one full cycle every 3 seconds. A second can be estimated by counting at a moderate rate, —one—two—three—four—five—six.

TREATMENT OF A CHOKING BABY

The Procedure.—

1. Pick up the child and invert it at an angle of at least 45°.
2. If there is a tendency for the offending matter to stick in the

Fig. 39.—To remove throat obstructions, proper method.



(Shown only to condemn.)
Fig. 40.—To remove throat obstructions, faulty technic.

pharynx, quickly throw a piece of gauze or a clean handkerchief around the little finger (Fig. 39), and insert the finger into the pharynx and remove the obstruction.

3. If a few minutes coughing does not give relief, call a laryngologist and have the larynx examined with a mirror.

4. If the larynx proves to be clear, have a roentgenogram made. The reader is referred to Chevelier Jackon's epochal work "Bronchoscopy" for information on this immensely important subject.

INTUBATION

In experienced hands, this process is superior to a tracheotomy for the relief of dyspnea in diphtheria of the laryngeal type. It is an operation that demands skill and practice.

Fig. 41.—O'Dwyer intubation set.

The Procedure.—

1. Wrap the child in a sheet restraining the arms and legs.

2. Place the patient on his back on a table with the head a trifle extended and have an assistant steady the head with his hands.

3. Attach to the introducer a tube of appropriate size, selected according to the age of the child. Be guided in this choice by the gauge that comes with the set. Loop a silk cord through the hole in the upper margin of the tube.

4. Insert a mouth gag and open the jaws widely.

5. Pass the index finger of the left hand to the back of the pharynx and bring it forward until the tip of the cricoid is felt.

Crowd the finger well to the patient's right pharynx leaving the middle line of the opening of the larynx free.

6. Pass the tube along the radial side of the index finger and direct the tip of the tube into the opening of the larynx by elevating the handle of the introducer. This part of the operation requires speed. If it is not promptly successful, withdraw the finger and make another attempt.

7. After the tube is passed into the larynx, touch the thumb piece on the handle of the introducer, release the tube, and by holding the tip of the finger against the margin of the tube, remove the introducer.

8. After a few moments, when it is certain that the tube is in proper position (as is evidenced by a coughing attack followed by a relief of the dyspnea), the index finger is removed, the mouth-gag is taken out and the silk thread attached to the tube is brought out of the corner of the mouth and fastened to the face with adhesive plaster. If an intelligent nurse is in attendance, it may be safe to remove the thread by cutting it in two while the index finger is still in the pharynx to prevent the tube from coming out. There is no danger of the tube's going on down into the larynx but it may be coughed out and swallowed. This will do no harm, as the tube readily passes with the bowel. If the cord is left in position, the child's arms must be carefully restrained or it will pull the tube out. There is always the danger, however, if the cord is removed, that the tube may become plugged up with secretions and if no experienced attendant is present, grave results may ensue.

9. If the tube is coughed out, replace it with another of larger diameter if possible, as soon as respiratory embarrassment becomes evident.

10. Leave the tube in position from 2 to 7 days as is required.

11. To remove the tube, put the child in the same position as was used when the tube was inserted.

12. Use the same technic as was used in inserting the tube. Guide the tip of the extubator into the opening in the tube, separate the jaws of the instrument by making pressure on the lever in the handle. Withdraw the tube by making a semicircular downward sweeping movement with the hand holding the extubator.

13. Observe the patient for several hours and at the first sign of dyspnea, reinsert the tube. It is important to have an intubation set sterile and ready for immediate use in the emergency.

OPEN TREATMENT FOR TREATING EXCORIATED BUTTOCKS

The Procedure.—

1. Obtain a cradle (Fig. 42) of wood or metal about 12 or 14 inches high and 2 to 2½ feet long.

Fig. 42.—Treatment excoriated buttocks.

2. Place rubber sheeting on bed, cover with a folded sheet, and place a pad of diapers over the sheet.

3. Place the patient in the knee-chest position on the pad with its knees in such a position that the pad will catch the excretions.

4. Adjust the cradle over the child.

5. Cover the cradle with bed clothing and leave the child's head emerging from the cover at the end of the cradle.

6. Keep the interior of the cradle warm with hot water bags or hot bricks; or suspend from the roof of the cradle, an electric light protected by a wire cage, such as is used in automobile repair shops, or an electric heating pad.

7. When the patient is soiled, cleanse him with light petrolatum oil; use no water on buttocks.

8. Dust the buttocks twice a day with a dusting powder composed of thymol di-iodid, 1 dram to zinc stearate, 1 ounce.

METHOD TO PREVENT FACE SCRATCHING

The Procedure.—

1. Cut a cardboard mailing tube into two lengths of 4 or 5 inches each.

Fig. 43.—Arm extension to prevent scratching.

2. Draw the tube over the child's arm up to the axilla on the outside of its undershirt. Roll the cuff of the undershirt back onto the lower end of the tube and fasten it in that position with a bandage if necessary. This precaution prevents the tube from falling off. (Fig. 43.) This method allows free movement of

the arms from the shoulders but prevents the infant from getting its hands to its face. Failing a mailing tube, any piece of cardboard or corrugated board will serve the purpose. Tongue depressors made of wood and sewed between two layers of cloth also make an acceptable restraining apparatus.

3. If there is a tendency for the child to scratch its face with its shoulders while the hands are so restrained, it may be necessary to replace this apparatus with a gown with sleeves about three inches longer than the infant's arms. The end of the long sleeve should then be pinned to the lower part of the diaper.

THE FACE MASK

1. Take a tube of stockinet 8 or 10 inches long, sew up one end and pull it on over the child's head. The top of a white cotton stocking may be used in place of the stockinet if desired.

Fig. 44.—The face mask.

2. On the stockinet, mark with ink the position of the eyes, nose and mouth, while the stockinet is over the child's head.

3. Remove the stockinet and cut holes at the points indicated by the ink marks. Take care to cut the holes very small as the

fabric stretches when it is put back on the patient's head and the holes may be too large. (Fig. 44.)

4. Soak the mask in melted yellow petrolatum. It should be remembered that petrolatum is irritating to some children's skins; for such patients a vegetable oil may be tried. The soaking up of the mask with oil will prevent the fabric from absorbing the medicament applied to the skin.

5. Apply the desired ointment to the face and replace the mask.

6. Remove the mask for laundry every day and apply a fresh one.

7. Launder the mask with a mild soap and rinse it at least 3 times with clear water.

VACCINATION

Equipment.—

1. Vaccine in capillary tubes.
2. Scarifier of the chisel type, a needle or a scalpel.
3. Green soap.
4. Alcohol, 50 per cent.
5. Sterile dressings.
6. Adhesive plaster.

The Procedure.—

1. Cleanse the area carefully with green soap solution and allow to dry spontaneously. The application of iodin or other strong antiseptics may interfere with the action of the vaccine; there can be no objection to the application of 50 per cent alcohol, however, if it is allowed to dry thoroughly.

2. Flame or boil the scarifier.

3. Wipe off the capillary tube with alcohol and allow it to dry. Push the capillary tube through the rubber bulb until the sealed end emerges from the top of the bulb. Break off both tips with flamed forceps. Draw the tube down until the broken upper end lies within the bulb.

4. Make the skin of the selected area tense by grasping the arm or the leg, if the latter be chosen, with the hand partly encircling the extremity.

5. Squeeze a drop of serum from the capillary tube on the area and make scarification through it with a quick turn of the chisel scarifier (or sterile needle may be employed). Or two strokes, $\frac{1}{16}$ inch long may be made with a needle. These lines should intersect and a drop of vaccine be placed thereon.

6. Allow the serum to dry spontaneously.

7. Apply a square of sterile gauze and fasten it into place with adhesive strips. The adhesive strips should not entirely encircle the arm; so placed, they tend to cause congestion and sloughing. Avoid the celluloid shield; it forms an ideal culture chamber for infective organisms.

When a formerly vaccinated patient is revaccinated, inspect the inoculation site 24 and 48 hours after inoculation. The appearance of an immunity reaction at this time indicates that protection from the earlier inoculation is still effective.

VON PIRQUET TEST

Equipment.—

1. Tuberculin, human.
2. Tuberculin, bovine.
3. Scarifier (chisel type), or needle.
4. Alcohol lamp or gas burner.
5. Green soap.

Fig. 45.—Von Pirquet test.

6. Alcohol, 50 per cent.
7. Sterile cotton or gauze.
8. Platinum loops.

The Site.—

1. Flexor surface of the forearm. (Fig. 45.)
2. Any smooth surface of the body. Forearm preferable.

The Procedure.—

1. Cleanse area with green soap solution; apply alcohol and allow to dry.

2. Flame the scarifier and allow it to cool.

3. Make scarifications. If the chisel scarifier is used, make 3 slight circular abrasions about 1 inch apart on the flexor surface of the forearm. This is done by giving the chisel a quick turn between the thumb and finger. If the needle is used, a linear scratch 3 inches long, just through the outer layers of the skin, is made across the area selected. (If preferred the abrasion may be made through a small drop of tuberculin.)

4. Apply the tuberculin with sterile probe or platinum loop. If tuberculin in capillary tubes is used, the tubes are opened in the same way as described in the method of vaccination. A small drop of tuberculin is all that is necessary. Leave one abrasion uninoculated for a control. Inoculate in some given order easily remembered. For example, from above downward.

O Human
O Control
O Bovine

5. If the scratch method is used, apply the tuberculins to the scratch mark in positions about 1 inch apart.

6. Allow the tuberculin to dry. A dressing is not required.

7. Make readings in 24, 48 and 72 hours. A positive reaction consists of a reddened area from $\frac{1}{8}$ to $\frac{1}{2}$ inch in diameter. The normal reaction to the trauma can be read on the control.

TECHNIC OF SCHICK TEST

1. Clean flexor surface of forearm with alcohol or ether.

2. Inject $\frac{1}{10}$ c.c. of diluted diphtheria toxin *into* the layers of the skin. Use sharp needle.

3. Control on other arm by using same site and amount of injection, but with the toxin heated to 75 degrees for five minutes.

4. Positive reaction, appears in twenty-four to thirty-six hours, and is characterized by circumscribed area of redness and infiltration. Reaches height by fourth day, then fades leaving brownish pigmented spot, which later scales.

5. Negative reaction. Practically no sign of injection in 24 hours.

6. Pseudoreaction. (A protein reaction) much more rapid in appearance, namely, 12 to 18 hours, fades in from 3 to 4 days, leaving slight brownish pigmentation without scaling.

7. State Boards of Health usually furnish titrated toxin with sufficient sterile salt solution to perform 50 tests when diluted. The toxin must be used at once after dilution.

TREATMENT OF UMBILICAL HERNIA

The Procedure.—

1. Cut a strip of good quality adhesive plaster 1½ inches wide and 6 or 7 inches long.
2. Lay the baby on its back on the mother's knee or on a table.
3. Be seated to the left of the baby.
4. Dust aristol or bismuth-formic-iodid onto the umbilicus.

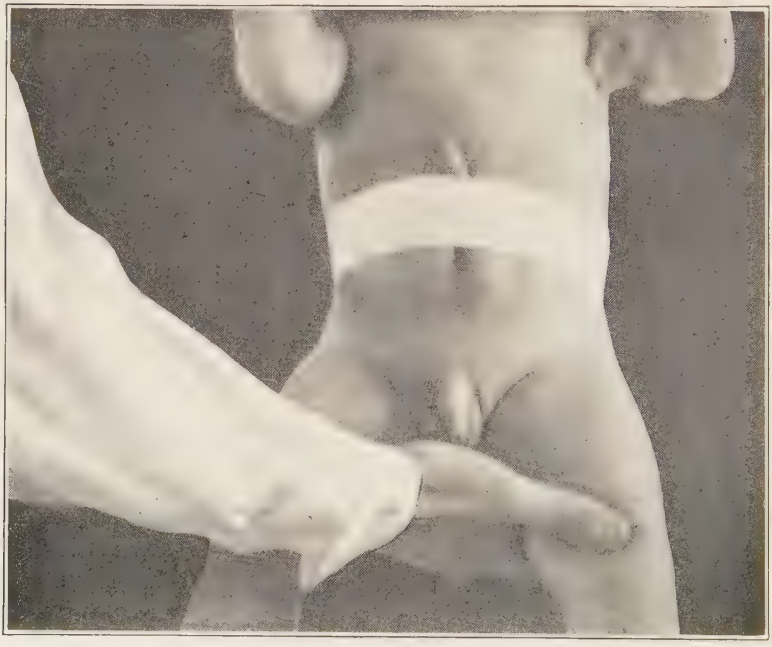


Fig. 46.—Treatment umbilical hernia.

5. Apply one-half of the strip of adhesive plaster firmly to the right side of the abdomen at a level with the umbilicus. Make traction on the free end of the strip with the right hand. With the index finger of the left hand, reduce the hernia. Hold it in reduction by maintaining a gentle pressure with the finger over the umbilicus.

With the finger still in this position, grasp a fold of abdominal wall of the left side of the abdomen between the thumb and index finger and bring this fold toward the median line. Withdraw the index finger from the umbilicus and simultaneously with firm traction, draw the strip of adhesive plaster over the umbilicus and make it adhere to the left side of the abdominal wall. The center of the adhesive plaster should be over a groove occupying a position between two vertical folds of skin. The reduced hernia should lie at the bottom of the groove. (Fig. 46.)

6. Leave the plaster in place until it loosens spontaneously. With a daily bath, this loosening will occur in 10 days or 2 weeks. In removing plaster, do not use benzin. Oil will loosen the muslin from the plaster without irritating the skin.

7. Dust the plaster adhering to the skin with talcum, rub it gently, wash with soap and water and the adherent plaster mass will come away clean.

8. In reapplying the plaster, cross the umbilicus at a different angle in order to prevent skin irritation.

TREATMENT OF INGUINAL HERNIA

The Procedure.—

1. Attempt to reduce the hernia with very gentle infolding manipulation of the tumor.

2. If any difficulty is encountered, give the patient a hot bath to relax the tissues, and have an assistant elevate the pelvis and allow the intestines and omentum to fall away, creating traction from the inside, on the sac contents. If the child struggles, this maneuver will not be effective as the muscular rigidity produced will only tighten the ring. If, with gentle manipulation, it is impossible to reduce the hernia, call a surgeon at once.

3. If the hernia is reduced, such reduction may be maintained by the application of a yarn "truss." (Fig. 47.) Mechanical trusses are of little value on infants and they are uncomfortable and may be dangerous by the production of too much pressure.

4. To make a yarn "truss," take a skein of soft knitting wool, known as Saxony. From it, select strands enough to make a smaller skein, appropriate to the size of the infant.

5. Sever the loop of the skein and tie the free ends for convenience in handling.

6. Make the first tie of a surgeon's knot, leaving a loop large enough to insert the leg of the same side as the hernia. The knot

should be tied so that there is a long and a short free end to the skein of worsted.

7. Slip the loop over the leg and fit it snugly around the thigh and in the groin. Have the knot just over the hernial opening. Complete the surgeon's knot, loosely tied.

8. See that the hernial sac is empty.

9. Draw the knot tightly and apply it snugly over the hernial opening. Turn the knot over.

Fig. 47.—Treatment of inguinal hernia.

10. Carry the right short free end of the skein across the knot to the *left* of the abdomen and around the body just below the brim of the pelvis.

11. Carry the left long free end of the skein across the knot to the *right* of the body just below the right superior spine, make firm traction and tie a restraining knot.

12. Several skeins should be provided that the truss may be changed when soiled.

METHOD OF TREATING ENURESIS

The following printed slip is given the mother.

Allow no fluid after —— p.m.
Take the child up at —— o'clock and again every —— hours during the night and take him to the toilet. Turn the lights on and be sure he is thoroughly awake. (Wash his face with cold water if necessary to waken him). This step is very important. After he knows where he is and what he is about, allow him to urinate.

Keep the foot of the bed elevated about six inches. (Wooden blocks under the foot posts will accomplish this purpose).

If the child can write, have him write each day on a piece of paper, "I wet the bed last night" or "I did not wet the bed last night." If he cannot write, have him make a mark, (a cross or a circle) after the days of the week. Have the child return this paper to this office on the day of your next appointment.

Your whole-hearted and patient cooperation is a necessity in this treatment; otherwise it cannot succeed.

NOTE: Gold and silver stars similar to those used by kindergarten teachers may be employed to stimulate interest on the part of the patient.

MANUAL EXPRESSION OF MILK

This way of emptying the breast or of procuring milk is superior to the breast pump method. In addition to emptying the breast more effectively, it stimulates the production of milk by massage. The mother can be readily taught the maneuvers and after a little practice, she becomes quite expert. The following directions should be given her:

The Procedure.—

1. Be seated comfortably near a low table.

2. Place a boiled container on the table so that the upper margin of the vessel comes just under the breast. (Fig. 48.)

3. Place the palm of the left hand either below or above the breast and exert gentle pressure, thus forcing the milk through the ducts toward the nipple.

4. With the balls of the thumb and index and middle fingers of the right hand, make a sweeping motion from the outer margin of the areola toward the base of the nipple. This motion should force the milk out of the nipple. Do not press on the nipple itself. Avoid painful manipulation. If you do not get the milk at first, keep on trying for a few minutes and you

will succeed. Nervousness, the novelty of the situation, or fear, may interfere with the process for a few minutes but a little patience and effort will accomplish results.

Fig. 48.—Manual expression of milk.

If the patient is in bed, she should be turned on her side and the container placed under the nipple. If it is desired to have a nurse do the manipulation, the same maneuvers are to be used.

It is well to have the nurse stand in front of the patient and a little to one side.

FEEDING WITH A BRECK FEEDER

The Procedure.—

1. Lay the child on its side.

2. Tuck a soft towel under its chin to collect any overflow of milk and thus prevent wetting of its clothing.

3. Fill the sterilized Breck feeder (Fig. 49) with the feeding

Fig. 49.—Breck feeder.

solution at body temperature. A rubber tipped medicine dropper may be used instead. (Fig. 50.)

4. Place the nipple in the infant's mouth and squeeze on the bulb just sufficiently to express the milk through the nipple no faster than the child can swallow it.

5. As the milk is taken, air enters the feeder sometimes with difficulty, especially if the hole in the nipple is small. It may be

Fig. 50.—Rubber tipped medicine dropper.

necessary from time to time to tip up the nipple and allow the milk to run back into the bulb end of the feeder, and to raise the edge of the nipple slightly in order that air may enter.

METHOD OF NURSING AN INFANT

The Procedure.—

1. Have the mother hold the child in a comfortable position with one knee elevated about 10 inches. (Fig. 51.)

2. With the index and middle finger of the hand of the opposite side from which the infant is nursing, the mother retracts the fatty portion of the mamma just above the areola, thus preventing the gland from obstructing the nostrils.

FEEDING THE THICK FORMULA

The Procedure.—

1. Divide the warmed feeding into two parts and place in tumblers.

2. Put tumblers in a water-bath in order to keep them warm.

3. Take a large rubber nipple (base about 2 inches in diameter), (breast type of rubber nipple) and with the point of a sharp

Fig. 51.—Nursing infant, proper method.

knife, make a $\frac{1}{8}$ inch slit in the extreme end of the teat. Boil the nipple.

4. Place the patient on its back. (Fig. 52.)

5. Spoon the feeding into the nipple from the tumblers. While one half is being given, the other is being kept warm in the water-

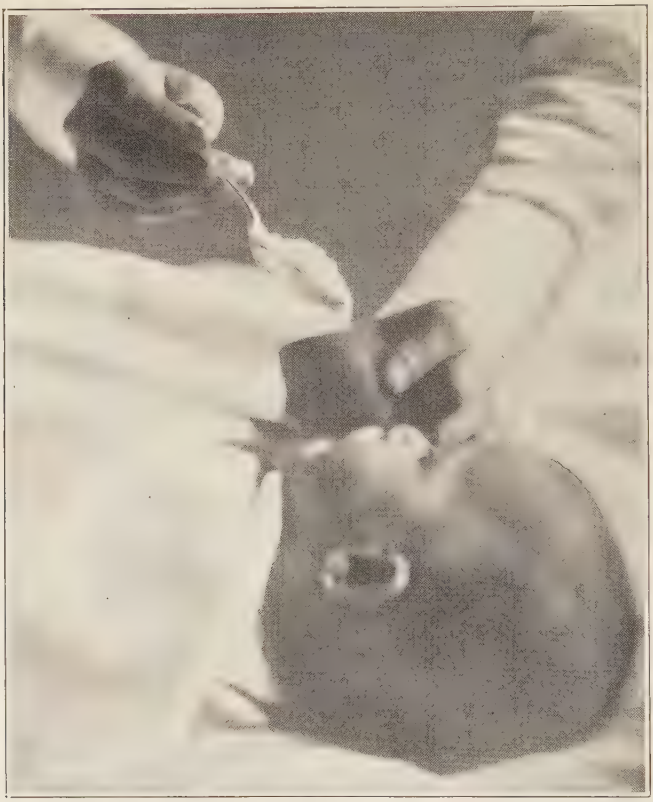


Fig. 52.—Feeding thick formula.

bath. When properly made and given at proper temperature, the food should be a viscid mass that will just flow from the spoon.

6. After the infant has taken the feeding, it is well to place it in an inclined position with the head high.

HELIOTHERAPY

The Procedure.—

1. *If the abdomen is to be treated,* lay the child on its back on a couch or covered table. Expose one-half of the abdomen to the direct rays of the sun without the interposition of any shading device whatsoever. Protect the eyes from the glare of the sun by goggles. Begin with a 5-minute exposure.

2. Increase the time of exposure 2 to 5 minutes each day. Increase the area exposed little by little, until the entire abdomen and lower chest is being exposed. During cold weather, the ex-

posure room should be heated sufficiently warm to keep the patient comfortable.

3. Gradually increase the exposures until a maximum of 4 or 5 hours daily has been attained.

4. As the time of exposure is being increased, the area of exposure should also be increased until the child is taking the exposures entirely naked.

5. If a chronic suppurating area is being exposed, the condition of the wound will determine to a large extent the time of exposure. In some instances, it may be necessary to discontinue the exposures for a few days or to lessen the exposure time.

Heliotherapy is coming to be recognized more and more as a potent therapeutic weapon, particularly in conditions where chronic disorders are encountered. It has been shown that the metabolism rate is markedly increased when heliotherapy is employed. In tuberculosis, particularly of bone, joint or gland, in rickets and in convalescence after acute infections, it is particularly valuable.

The technic consists of a process of gradual inurement. This, of course, will vary with different localities as well as with differences of complexion. After a few days or weeks, the child becomes able to stay in the direct sunshine for the entire day, clothed only in a low-cut bathing suit or cotton slip.

FLUOROSCOPY IN INFANCY

The Procedure.—

1. Remove all the clothing except the diaper and stockings.

2. Enter fluoroscopy room, darken except a small ruby lamp.

3. Allow a few minutes' time to elapse to accommodate vision.

4. Have the assistant hold the child in an upright position, behind the fluoroscopic screen and support it with one hand held under its buttocks and the other under its chin. This maneuver is necessary in order that the shadow of the assistant's hands may not interfere with the chest image.

5. Turn off the red light. If a troposcopic table is available, the child may be placed on its back or abdomen.

6. Turn on the current lighting the tube and make observations. Note the expansion of the diaphragm, the action, position and contour of the heart and the presence of abnormal shadows in the chest. If the gastrointestinal system is under investigation and a barium meal has been given, note the size, shape, motility and outline of the stomach, and the presence or absence of the duo-

denal cap. Note the size, position and contour of the barium shadow, if any be present, in the intestine or colon. If an opaque enema has been given, note the size, position and contour of the rectum, sigmoid and colon. The observation can continue for so much as a half-hour or more without injury to the patient.

THE RADIOGRAM

Of the Head.—

A radiogram of the head of an infant may supply evidence of some value in the study of intracranial disease. Whenever a **tumor** of the brain is suspected, it is in order to take an x-ray picture of the skull, although negative roentgenographic evidence will be of little value. However, if there be a **pituitary tumor,** the sella turcica may give evidence of diagnostic importance. In a case of **hydrocephalus,** after fluid is withdrawn from the ventricles and they are filled with air, a radiogram may be made which will give accurate information about the distention of the ventricles and their communications with each other and about the degree to which the brain tissues have been compressed.

A radiogram of the skull very clearly shows alterations in the bones of the vertex. **Craniotabes, improper ossification, separation of sutures, increase in the area of the fontanels** and **exostoses** all clearly show on the plate. Injuries to the skull are rarely productive of **fissured fractures** of the vertex. **Fractures of the base** as a rule are not visible on the radiographic plate. **Depressed fractures** of the vertex somewhat analogous to the green stick fractures of the long bones occur, and are readily revealed by the x-ray. A study of the shadows at the base of the infant's skull shows very little. It is occasionally possible to confirm the other clinical evidence of inflammation in the **mastoid cells.** In those cases in which the pus of mastoid inflammation comes forward through the anterior wall of the external auditory canal, to lie under the parotid gland and along the zygoma, a radiogram is a distinct aid to diagnosis.

Of the Chest.—

There is no doubt of the value of a well taken plate as an aid to diagnosis of disease of the infant chest. Immediately after birth, a picture may confirm the presence of an **atelectasis** or **congenital heart disease.** A little later in life, an **enlarged thymus** may be suspected and its presence can be proved by the radiogram. In that group of **pneumonias** in which physical signs fail

for some days, the evidence afforded by a radiogram may be conclusive. Such cases are not at all rare during the first year of life. If the patient be in a hospital, it is often illuminating and interesting to follow the course of the shadow in the lung from day to day during the course of the disease. **Empyema** following pneumonia is clinically difficult of determination until there is a considerable accumulation of pus. A study of the x-ray plate in such a case will often make it possible to discover smaller amounts of pus while it is free in the pleural cavity or as a localized empyema.

On rare occasions in early infancy, enlargement of the **mediastinal** and **bronchial glands** may become a source of disturbance in the chest. The enlargement of these glands and their character and location in the chest produces shadows on the plate that are susceptible of ready and accurate interpretation. On rare occasions, the **adult type of tuberculosis** is revealed in one or the other of the lobes of an infant's lung, but interpretation under these circumstances must be very conservative, as subacute, nontuberculous infiltration occurs which it is almost impossible to differentiate. Deepening of the shadows about the hilus of the lung and those produced by the peribronchial tissues radiating out into the substance of the lung, may mean a **mediastinal gland tuberculosis** with peribronchial tuberculous infiltration; but this interpretation should be made with the greatest caution because experience with x-ray plates will show that the lungs of perfectly healthy infants will throw lung-root and peribronchial shadows which are exceedingly difficult to evaluate. Certainly, diagnosis of tuberculosis in a child under 2 years of age should not be made on the roentgenogram alone, excepting possibly **tuberculosis of the miliary type.** In such a case, where the milia are widespread in the lung, the plate is typical. The tiny masses throw no very distinct shadow, but by their numbers, they interfere with the passage of the rays in such a way that the finished picture has an appearance well described as "mossy."

Rarely, **tumors** or **abscesses** of the lung may be shadowed on the plate. Opaque **foreign bodies** which have been aspirated, are readily seen and sometimes the radiogram is of value in attempts to locate a foreign body which is not opaque; such a body, perhaps a bean or a pea, may lodge and occlude a bronchus and the resulting area of collapse and consolidation may cast a shadow that is visible on the plate.

The **normal heart shadow** in infancy should appear on the plate as a conical, pear-shaped opacity of moderate size. The lower margin blends with the shadow cast by the liver and diaphragm and the upper runs into the shadow of the great vessels and of the thymus gland. The **thymus gland** is always apparent to a greater or less degree during the first year, but normally it never approaches in size the normal limits of the heart shadow.

Alterations in position of the heart may occur. Rarely, the heart may be transposed and occupy a position on the right side analogous to that of the normal heart on the left side. These transpositions may be mistaken for pathological displacements of the heart. **The heart may be pushed or drawn laterally** away from its normal position, or it may be crowded upward by accumulations in the subphrenic space.

The heart shadow may be greatly enlarged in size and its contour altered. An approximately round opacity, the "cannon ball type" of heart shadow is indicative of a **congenital defect.** The roundness is a result of dilatation and hypertrophy of the right side of the heart, most often the result of a **pulmonary stenosis or atresia.** The appearance of a cardiac opacity somewhat larger than normal but with a shadow superimposed above and to the right, occurs often when the child has the symptoms of an **open ductus arteriosus.** The x-ray appearance of **pericardial effusion** is that of a fairly symmetrical enlargement of sac-like outlines running well up onto the conus arteriosus.

Of the Abdomen.—

The x-ray is of little aid in the study of gastrointestinal conditions during infancy except when there is a question of **obstruction** in the tract. Here an opaque meal may make the site of the obstruction evident. It is rare that an experienced diagnostician, with the full data of history and examination at hand, in a case of **pyloric obstruction,** gains any new information from the knowledge acquired by a study of the radiographic plate or of gastrointestinal movements by aid of the fluoroscope. These observations are of confirmatory value only. There is no question that in certain cases of pyloric obstruction, the observations of the opaque meal through the fluoroscope may mislead the observer. Under the writers' observation in at least two instances where the clinical evidence warranted the diagnosis of hypertrophic obstruction at the pylorus, the opaque meal left the stomach and passed through the bowel promptly. When because of

persistence of symptoms, these cases were brought to operation, the perfectly typical, large, hypertrophic mass of pyloric muscle was found and incised, with the result that the symptoms immediately subsided.

The use of the opaque meal during infancy followed by fluoroscopic observations will often reveal the site of **partial or complete obstruction** of the bowel. Snow, of Buffalo, was first to suggest the use of the bismuth enema as an aid to the diagnosis of **intussusception.** A plate taken in anterior projection but without the ingestion of an opaque meal, may show the intussusception tumor outlined by gas accumulation in the gut. Most often the history and manual examination renders the diagnosis of intussusception clear but these methods add a visual factor to the other means we have of determining this type of obstruction. The fluoroscope may be used as an adjunct to the Hirschsprung method of hydrostatic reposition of the intussuscepted intestine. The operation may be performed under the fluoroscope and a barium suspension may be flowed into the rectum and be made to produce pressure against the descending knuckle and the unfolding of the intestine may be observed.

A good plate of the abdomen will demonstrate the liver and spleen and sometimes the kidneys; usually the colon will contain enough gas to outline its course if not the detail of its wall. Such shadows must not be mistaken for **tumor masses.** Tumor masses in the abdomen sometimes may be located by distending the colon with air or oxygen and taking an x-ray picture. The position of the large intestine as it shows on the plate in relation to the shadow of the tumor mass may be of service in diagnosing the site from which the tumor springs. This plan is of much value when the tumor is of renal origin. Nitrogen and oxygen each has been injected into the peritoneum before making radiograms of suspected tumor masses. Apparently the method is one of great promise.

Of the Spine and Extremities.—

The radiogram is invaluable in diagnosing lesions of the long bones. Clinical evidence of beginning **caries of the spine (Pott's disease),** may be confirmed by a rarefaction and roughening of the bodies of the vertebræ, but Pott's disease should be under treatment long before there is any radiographic evidence. Persistent abdominal pain in a child after its first year, together with an unwillingness to move its body freely, should call for

an x-ray picture. In most cases of **achondroplasia**, a roentgenogram will show lumbar vertebræ of anomalous development.

Radiograms of the long bones may show **hemorrhages** and **suppuration** about the epiphyses and under the periosteum. **Epiphyseal separation** following hemorrhage, suppuration or injury will also be observable. Shadows cast by erosions and thinning of the bones during the course of an osteomyelitis are diagnostic. It should not be forgotten that during the early stages of an osteomyelitis, the x-ray is of no diagnostic value, as the mere accumulation of pus in the medullary canal or in the bony trabeculæ does not cast a shadow on the plate. Later in the course of the disease, granulation tissue invades the bone and rarefies it, giving definite evidence on the roentgenogram of the extent of the process.

The radiographic appearance of the epiphyseal cartilages and the diaphyses peculiar to **rickets** is characteristic. A widening of the transverse dimension of the epiphyseal line which produces a flaring of the end of the diaphysis occurs, and there is a thickening of the cortex on the concave side of the long bone which has undergone some degree of bowing.

The x-ray picture of the long bones of a well developed case of **scurvy** is characterized by subperiosteal hemorrhages about the epiphyseal cartilages, the diaphyses and the shaft of the long bones. Even small hemorrhages are clearly visible and after a few weeks, the visibility is increased by a deposition of a layer of new bone. In advanced cases, a peculiar, transverse line particularly seen along the upper diaphyseal margins of the tibiæ and fibulæ, known as the "white line," is often recognizable.

The characteristic x-ray appearance of a **tuberculous process** in bone is the extreme destruction with a minimum of repair. Tuberculosis of the long bones may be divided into that of the shaft and that of the joint. **Joint tuberculosis** may originate as a synovial process, in which case the first radiographic evidence is a diminution in the spacing between the adjacent bone shadows resulting from erosions of the articular cartilages. The only evidence which can possibly precede this finding is the occasional visibility of thickening in the soft tissues or of fluid in the synovial sac. Subsequent spreading of the synovial process will produce a visible erosion into the bone on one or both sides of the joint space. When the process originates in the bone near a joint, it is primarily a tuberculous osteomyelitis and a focus of rarefac-

tion will be demonstrable, often before involvement of the joint occurs. After involvement of the joint has occurred, the process cannot be differentiated from the one in which the original focus was in the joint. **Tuberculosis of the shaft** characteristically begins in the diaphysis near the epiphyseal line, by the formation of rarefied areas which extend down the shaft and upward across the epiphyseal line. There is little evidence of sclerosis or periosteal reaction such as is found in osteomyelitis and to a less extent in lues. The commonest sites for bone tuberculosis are hips, spine, shoulders, tarsi and phalanges.

Syphilis of bone is uncommon during the first two years of life, but when it does occur, it is most often found affecting the tarsus and metatarsus, carpus and metacarpus and the phalanges of the hands and feet. The radiographic evidence of lues and tuberculosis, especially of these smaller bones, is very like in appearance. In favor of lues is multiplicity of lesions with a tendency to symmetrical, bilateral distribution. Marked evidence of periostitis with raising of the periosteum and proliferation of osteoblasts is further evidence in favor of the luetic origin of the bone disease. If the lesions are exclusively in the shafts of the bone, the epiphyses are unaffected. This fact is also to be taken as suggestive evidence of bone syphilis. When the long bones are affected, it is most unusual to have an epiphyseal damage. This is quite contrary to the rule that holds when the tubercle bacillus is the infecting agent. Scattered spotting of the epiphyseal shadows is an evidence of early tuberculosis and speaks against lues. There is one form of bone lues, however, in which these appearances will not be found, and that is when the clinical picture is one of **luetic pseudopalsy.** In this manifestation of spirochete activity, the incidence of the damage is always at or near the epiphyses and the radiographic findings are those of an epiphysitis.

Separation of epiphyses is an accident not at all uncommon in the life of the runabout infant. The lower epiphysis of the tibia, the lower radial epiphysis and the epiphyses of the elbow are those commonest affected. The partial separation shows clearly on the radiographic plate and it is the only means we have of determining the lesion.

Fractures of the long bones are fairly common. They are usually of the green stick type during infancy, and they are readily shown on the radiogram.

In examining a radiographic plate of an infant, it is necessary that the observer keep clearly in mind the roentgenoscopic features peculiar to infancy. A knowledge of these peculiarities, particularly those of the long bones and of the contents of the chest cage, will go far in assisting the physician in the recognition of abnormalities.

We are indebted to W. Edward Chamberlain for the following epitome of the normal centers of ossification evident during infancy and for a description of the appearance of the normal chest plate of the young child:

At birth, the **diaphyses** of all the long bones are well ossified, and considerable portions of such bones as the ischium, ilium, pubis, scapula, vertebral bodies and skull bones have also undergone ossification. But the only **epiphyses** which have developed osseous centers are the femoral and tibial epiphyses at the knee joint. The carpal bones are entirely cartilaginous, as are most of the tarsals, but the os calcis and astragalus display well developed centers, and the center in the cuboid normally develops just before or just after birth.

Very soon after birth, we should find osseous centers developing in the os magnum and unciform bones of the carpus, and at about the sixth month, the upper femoral and upper humeral epiphyses develop centers, closely followed by the lower epiphyses of the tibia.

At the age of one year, the average normal infant will have osseous centers in 2 bones of the carpus, (os magnum and unciform); in four bones of the tarsus, (os calcis, astragalus, cuboid and external cuneiform); and in the following more important epiphyses: lower femoral, upper tibial, femoral head, head of the humerus, lower tibial and frequently the capitellum.

During the second year, we should have added the following centers: the capitellum (if it has not already appeared), the coracoid process of the scapula, the lower radial epiphysis, and the lower fibular epiphysis. These may be considered rather as a minimum, for it is within normal limits for the normal child to exhibit, in addition to the above, such centers as cuneiform in carpus, internal and middle cuneiforms in tarsus, tuberosities of humerus, and beginnings of patella. Less often, a few of the epiphyses of metacarpals, metatarsals and phalanges may appear at this age.

The radiographic appearance of the **normal infant chest** is not constant. It varies not only with the normal anatomical pecu-

liarities of different children, but also with the technic used in the exposure of the x-ray plate.

If the child be placed face downward with chest upon plate and tube centered from above, the resulting "anterior projection" should show the following obvious and important details:

The **spine** shadow, ideally, should be straight. Practically, it is difficult to catch a lively infant with its spine actually straight. It should be noted that a curved spine shadow may account for many asymmetries. The **rib cage** should be symmetrical. This is in contrast with the narrowing in the "fetal type" found in many nutritional disturbances. An asymmetrical rib cage may be ignored when due to twisting of the child or to faulty centering of the x-ray tube. The shadows of the **scapulæ** usually lie outside those of the rib cage in an anterior projection, for the diverging rays pass between the scapulæ to reach the ribs. The **clavicles** are very high in position on the infant's chest plate, usually above the upper limits of the lung fields. If the plate is a true sagittal projection, the spine shadow will pass straight downward between the sternal ends of the clavicles. The **lung fields** are bounded mesially by the heart vessel shadow and the superior mediastinal shadow, below by the diaphragm shadow and above and laterally by the costal wall. The costodiaphragmatic angles are sharp and acute. The angles between the shadows of the heart and diaphragm are also acute. Projecting into the lung field from its mesial boundary is the **hilus** shadow, cast by the major bronchi, larger pulmonary vessels and more central lymph nodes. From the hilus shadow, the shadows of the bronchial tree branches and their accompanying vessels, may be traced outward in all directions. These shadows are most pronounced near the hilus, and they fade out about two-thirds of the distance from the hilus to the pleura, leaving an outer, clear zone, normally devoid of "lung markings." The heart vessel shadow normally occupies more of the left chest than of the right, and particularly hides part of the left hilus and descending bronchial tree branches, making the right appear more prominent. Normally the **interlobar fissures** are invisible, but the importance of keeping their positions in mind is obvious. Degrees of pleuritic thickening, insufficient to cast a shadow anywhere else, will be caught in profile here, and it will show as a thin, sharp streak across the lung field, from hilus to lateral chest wall. The **diaphragm** shadows are smoothly convex upward. Normally, the right is somewhat higher than the left, but in the infant, a relatively high diaphragm

shadow on one side may be normal when due, for example, to a distended stomach, or to the uneven play of muscles. The right diaphragm shadow is always very apparent, as the division between liver density and lung field is marked. But with a gas distended stomach or splenic flexure, the left diaphragm shadow may be represented only by a very thin curved line, especially difficult to make out if it underlies a rib. About two-thirds of the area of the heart vessel shadow lies to the left, and one-third to the right of the median line. The normal shape soon comes to be recognized easily. The size must be considered in relation to that of the child, as indicated by the long fields and rib cage. Careful measurements of the shadow's area are probably not more valuable than the general impression of an observer who is familiar with the appearance of the normal chest picture. The **superior mediastinal** shadow is composed of trachea, esophagus, great vessels, lymph nodes, and particularly of the thymus gland, and the shadow may normally be hidden behind the spine shadow. But on account of the relatively large thymus of the infant, the superior mediastinal shadow is frequently evident on one or both sides of the spine shadow, especially if projection is not exactly sagittal.

If the child be placed face upward on the x-ray plate, and the tube centered as in the anterior position, the result will be a "posterior projection" with the following differences from the anterior plate: The heart vessel shadow appears relatively larger, the ribs slope more steeply, the scapulæ and shoulder joints are more evident as the scapular shadows are no longer thrown outside the lung field, and detail in the spine and posterior portions of the ribs is better. It is important that true sagittal projection be made, otherwise, organs may appear displaced and among other things, the sternal ossification centers of the ribs may cast shadows into the lung field, closely resembling small coins or other foreign bodies.

HANDBAG NECESSITIES FOR PEDIATRIC PRACTICE

1. Stethoscope
2. Reflex hammer
3. Tape measure
4. Culture tubes
    a. Glucose agar
    b. Loeffler's blood serum.
5. Tongue depressors

6. Applicators
7. Cotton in spring bottom cotton holder
8. Mouth gag
9. Stomach tube
10. Soft rubber male catheter, size 9 French
11. Lumbar puncture needles
12. Clean, sterile test tubes
13. Adhesive plaster
14. Bandages
15. Dressings
16. Pocket surgical case
17. Hypodermic and extra needles
18. Head mirror or electric otoscope
19. Ear speculums
20. Flash light with extra battery
21. Ophthalmoscope
22. Intravenous needles
23. Glass slides with wood container
24. Hemocytometer pipettes
25. Talquist hemoglobin indicator book
26. Rubber gloves
27. Finger cots
28. Vaccination tubes
29. Tuberculin, human and bovine
30. Scarifier
31. Record book
32. Prescription blanks
33. Typewritten direction slips
34. Drugs
    - Solutions:
      - Alcohol
      - Iodin, 2%
      - Silver nitrate, 15%
      - Chloroform
    - Tablet triturates:
      - Ipecac and antimony, of each 1/100 grain
      - Calomel, 1/10 grain
    - Hypodermic tablets
      - Codein
      - Morphin
      - Heroin
      - Atropin
      - Apomorphin
      - Caffein sodium benzoate
    - Ampoules
      - Camphor (10%) in olive oil
      - Adrenalin, 1 to 3500
      - Atropin 1/200 gr. and adrenalin chloride solution 1/1000
      - Strophanthin

CHAPTER XXIII

FORMULAS AND RECIPES

MILK

Sterilization

With an Arnold Sterilizer:

The Equipment.—

1. Arnold Sterilizer.
2. Nursing bottles, required number.
3. Pitcher containing solution.
4. Funnel.
5. Cotton for plugs.

The Procedure.—

1. Pour the formula in the required number of bottles and plug with cotton, or cover with muslin squares.
2. Place bottles in a wire rack and put in sterilizer.
3. Start the flame under the sterilizer and after the water is boiling allow sterilization process to continue for 30 minutes. The Arnold Sterilizer holds the temperature at about 100° C.
4. Put the feedings in the ice box or cooler.

Without an Arnold Sterilizer:

The Equipment.—

1. Double boiler. (The ordinary kitchen double boiler, deep and narrow preferred.)
2. Nursing bottles, required number.
3. Funnel.
4. Pitcher containing formula.
5. Two inch squares of muslin.
6. Small rubber bands.

The Procedure.—

1. Pour the formula into the inner chamber of the boiler.
2. Put on direct flame and bring to vigorous boil. (This will prevent the formation of a skum on the surface.)
3. While this is being done, have the water in the outer chamber of the boiler heated.

4. Put the inner boiler into the outer boiler, set on the flame and allow to remain 10 minutes.

5. Add boiling water to make up for evaporation and divide the formula into the required number of feedings.

6. Cover the bottles with the small muslin squares which have been boiled and kept sterile and hold them into place with rubber bands.

7. Place the feedings in an ice box or cooler.

PASTEURIZATION

Freeman method of pasteurization.

The Equipment.—

1. Freeman pasteurizer.
2. Nursing bottles, required number.
3. Two inch squares of muslin.
4. Rubber bands.
5. Funnel.

The Procedure.—

1. Fill the bottles to the required amount with the feedings.

2. Fill the pail to the groove with water and raise to the boiling point.

3. Place the bottles containing the formula, corked with cotton plugs or covered with the muslin squares, into the cylindrical cups.

4. Pour sufficient water into the cups to surround the bottles.

5. Remove the pail from the flame, set it on a board or other nonconductor, and place the receptacle containing the bottles in it. In 10 minutes the formula is at a temperature of 155° F. The hot water will maintain this heat for 20 minutes.

6. In half hour, remove the bottles and put in cooler or ice box.

Home pasteurization.

The Equipment.—

1. Double boiler.
2. Bottles, required number.
3. Pitcher of formula.
4. Funnel.
5. Two inch muslin squares and rubber bands.

The Procedure.—

1. Place the milk in the inner boiler.

2. Place inner and outer boilers together and allow them to remain over a flame for 20 minutes.

3. Divide the formula into the required number of feedings. Fill bottles. Cover bottles with sterile muslin squares and hold these in place with rubber bands.

4. Cool rapidly in tepid water and then in cold water and put on ice or in cooler.

TO MAKE LACTIC ACID MILK

The Equipment.—

1. Culture of lactic acid bacilli (fluid preferred).
2. Large bowl.

Fig. 53.—Apparatus for pureeing vegetables.

3. Sieve. (Fig. 53.)
4. Wooden potato masher and a large fork.
5. Incubator or woolen blanket.
6. Fresh milk (1 quart).

The Procedure.—

1. Put lactic acid bacilli in solution with water.

2. Pour milk (boiled 3 minutes and cooled) into bowl, add the culture, stir thoroughly.

3. Put milk in milk bottle or clean jar and put in a warm place. An incubator is preferred. A water-bath with a rheostat is desirable. Failing these, the bottle may be wrapped in a small woolen blanket and kept in a warm room or on the plate rail of a stove. If the milk is kept at about body temperature for 12 hours, it will thicken very well. If at lower temperatures, 24 to 36 hours will be required.

4. After the milk has thickened, return it to the bowl, beat it thoroughly with a fork, and pass it through a fine wire sieve with to and fro movements of the potato masher. Repeat this process if necessary. Put on ice.

5. Save a few ounces of the soured mixture to be added to the next day's formula as a culture.

NOTE: Eight to 15 per cent of "Karo" syrup (brown) added to lactic acid milk constitutes the "Karo-Lac" extensively and very successfully used by Marriott.

TO MAKE PROTEIN-MILK (EIWEISSMILCH, ALBUMEN-MILK)

The Equipment.—

1. Junket tablets.
2. Bowl or pitcher.
3. Sieve. (Fig. 53.) Cheese-cloth for strainer.
4. Wooden potato masher.
5. Boiled water, two quarts, containing ¼ teaspoonful of salt.
6. Fresh milk. (1 quart.)
7. Lactated milk or good buttermilk. (1 pint.)

The Procedure.—

1. Heat the milk to body temperature.

2. Dissolve one junket tablet in a little warm water.

3. Stir dissolved tablet into warm milk.

4. Allow mixture to stand in a warm place for 20 to 30 minutes. The milk will thicken. This is junket.

5. Beat the curd of coagulated milk with a fork.

6. Spread 2 or 3 layers of cheese-cloth over the sieve and pour the divided curd onto the cheese-cloth.

7. Manipulate the cloth so that the fluid part of the milk runs through the sieve into a container. Allow to drain for half-hour. This filtrate is whey.

8. Pour the salt water, a little at a time, on the mass of curd and toss and twist the curd by manipulating the cloth as it lies over the sieve. Use the full two quarts. The salt water makes the curd less tough.

9. Wash the curd again with a pint of warm boiled water in order to remove the salt.

10. Turn the curd out onto the sieve.

11. Pour the lactated-milk or buttermilk over the curd, a little at a time, and work the mass through the sieve with a to and fro motion of the potato masher. The finest sieve that the mass can be passed through should be used. A little boiled warm water added to the buttermilk will facilitate the passage of the curd through the sieve.

12. Sweeten with saccharin or add any prescribed carbohydrate.

13. Divide into the required number of feedings, cover bottles with sterile muslin squares held in place with rubber bands. At feeding time do not warm the mixture above 160°.

TO MAKE PEPTONIZED MILK

The Equipment.—

1. Tube of peptonizing powder.
2. Bowl or pitcher.
3. Nursing bottles, funnel and small muslin squares and rubber bands.
4. Fresh milk.

The Procedure.—

1. Dissolve the contents of two peptonizing tubes in one ounce of cold water.

2. Pour quart of milk into bowl, add the peptonizing solution and stir thoroughly. Put the mixture in clean jar or bottle and set in a warm place for 20 minutes. The mixture should be held at body temperature. An incubator, a water-bath, a warm stove shelf or a warm blanket near a stove may be employed.

3. Make the prescribed formula, divide into the required number of feedings, cover the bottles with sterile muslin squares fastened into place with rubber bands.

4. Put bottles in ice box or cooler.

TO MAKE WHEY

(See protein milk making, page 591.)

TO MAKE JUNKET

(See protein milk making, page 591.)

TO MAKE MALT-SOUP FORMULAS

The Equipment.—

1. Malt-soup extract.
2. Flour, wheat or rye.
3. Salt.
4. Fresh Milk.
5. Double boiler.
6. Cup.
7. Pitcher.
8. Nursing bottles, small muslin squares, rubber bands.

Fig. 54.—Leveling a tablespoon.

The Procedure.—

1. Measure the prescribed amount of flour into a cup, make into a paste with cold water.

2. Put the prescribed amount of milk into the inner chamber of a double boiler and heat directly over a flame, stirring constantly.

3. Place the flour paste in a bowl, add a little of the hot milk, mix thoroughly and add this to the rest of the hot milk in the

boiler, stirring constantly. Allow the mixture to boil vigorously for 5 to 10 minutes.

4. Place the inner chamber of the boiler containing the mixture inside the outer chamber containing boiling water. Put on the lid and allow to boil for 35 minutes.

5. Remove the inner chamber and cool the mixture to blood heat by setting it in a cool place or in cold water.

6. While the milk is cooling, take the prescribed amount of malt-soup extract and dissolve in the prescribed amount of water at body temperature.

7. Add the malt-soup extract solution to the milk-flour mixture and allow to stand for 10 minutes.

8. Bring this mixture to a boil over a flame.

9. Add boiled water to compensate for what is lost in evaporation.

10. Divide into the prescribed number of feedings, cover bottles with small, sterile muslin squares and fasten them into place with rubber bands.

11. Put bottles in ice box or in cooler.

TO PREPARE POWDERED MILK

The Equipment.—

1. Glass graduate or large measuring glass.
2. Glass rod or large spoon.
3. Dry milk.
4. Boiling water.
5. Funnel.
6. Nursing bottles, small muslin squares, rubber bands.

The Procedure.—

1. Put boiling water in graduate.

2. Shake dry milk onto the surface of the boiling water, stirring constantly with glass rod or spoon.

3. Add any prescribed carbohydrate in solution.

4. Divide into required number of feedings, cover with squares of sterile muslin, fasten them into place with rubber bands.

5. Put in ice box or cooler.

6. When it is desired to feed infant, cap the bottle with nipple, heat to body temperature in water-bath, and shake thoroughly before giving to baby.

TO PREPARE BRADY'S MIXTURE
(Lactated Milk, Cereal and Maltose)

The Equipment.—

1. Pitcher.
2. Funnel.
3. Nursing bottles, small, sterile, muslin squares, rubber bands.
4. Bowl.
5. Sieve and potato masher.
6. 24 ounces of lactated milk. See page 590.
7. 8 ounces of barley gruel. See page 597.
8. 1 ounce Mellin's food.
9. ½ ounce granulated sugar.

The Procedure.—

1. Dissolve 1 ounce of Mellin's food and ½ ounce of granulated sugar in a little warm lactated milk. Add the remainder of 24 ounces of lactated milk and 8 ounces of barley gruel. Pass the mixture through a fine sieve with to and fro motions of the potato masher. Pass through sieve the second time if necessary to make a fine mixture.

2. Divide into the required number of feedings, cover the bottles with the muslin squares held in place with rubber bands. Put in ice box or cooler.

(Jules Brady uses this formula for young infants. It is valuable in many cases of difficult feeding.)

TO MAKE THICK FORMULA

The thick formula (concentrated cereal milk), originated by McClure and successfully adapted by Sauer, is of great value in treating vomiting babies with pyloric obstruction, and others suffering from malnutrition.

The Equipment.—

1. Double boiler.
2. Stirring spoon.
3. Covered enamel pan.
4. Farina or rice flour.
5. Prescribed milk formula.
6. Boiled water.

The Procedure.—

1. Mix the required amount of milk and water together; bring to a boil. Then sprinkle in slowly 8 level tablespoonfuls of the

farina to each 30 ounces of liquid and boil over a direct flame for 10 minutes, stirring continually. Then boil in a double cooker for 1½ hours. If rice flour is used, make into a paste with cold water before adding it to the boiling mixture. Use rice flour in the proportion of 10 level tablespoonfuls to each 30 ounces of liquid. After the mixture is cooked, add the prescribed amount of sugar.

EGGS

EGG-DRINK

Take the yolk of 1 egg; orange juice, 4 ounces; milk sugar, 2 heaping tablespoonfuls. Dissolve the milk sugar in 4 ounces of boiling water. Shake in a shaker or beat with an egg beater. (Bottled grape juice or loganberry juice or the juice of canned apricots or peaches may be used instead of the orange juice if desired.)

ALBUMEN-WATER

Take the whites of 2 eggs, stir into plain water, slightly salted or into barley water, 8 ounces. Strain through cheese-cloth or a fine sieve. Sweeten with a saccharin tablet if desired.

EGG-JUNKET

Make some plain junket. (See protein-milk, page 591.) Stir (do not beat) one egg into one-half pint of junket. Flavor to taste with vanilla or chocolate.

BOILED CUSTARD

Take one cup of milk, pinch of salt, yolk of one egg and 1 level tablespoonful of sugar. Bring the milk to the boiling point without actually boiling it; take it from the fire, have the egg and sugar beaten to a cream and pour the milk slowly over them, stirring all the time; replace on the fire in a double boiler and stir until the custard coats the spoon or is of a rich, creamy consistency. Turn into a cold dish and add vanilla as flavoring. Fruit may be puréed through a fine sieve and added to the custard.

BAKED CUSTARD

Prepare the mixture as for a boiled custard. Pour into cups and set the cups in basin of warm water. Bake in a moderately

hot oven. Just as the surface of the custards begins to brown, turn off the oven or open the oven door.

CREAMED EGG-YOLK

Put an egg in cold water, allow the water to come to a boil and boil for 20 minutes. Remove and drop into cold water. Turn out the yolk which should be powdery. Add a teaspoonful of butter to the yolk and rub into a smooth paste.

(Some children digest egg cooked in this way when they fail to digest it in other forms. The cream mixture may be added to milk formulas, broths, fruit purées or cereal feedings.

SOFT BOILED EGG

Put an egg in cold water and cook for one minute after it reaches the boiling point.

EGG WITH TOASTED BREAD CRUMBS

Place stale bread in a baking pan and put in the oven and let dry until brittle; roll with a rolling-pin into crumbs, after which, they may again be placed in the oven and slightly browned. Add a soft boiled egg or hard boiled, creamed yolk to a half-cup of crumbs softened with milk, and put into the oven until the mass is set.

CEREALS

CEREAL WATER

Cereals available:

Steel cut oatmeal.
Steam cooked oatmeal.
Farina or other wheat cereal.
Semolina.
Rye meal.
Corn meal.
Sago or tapioca.
Pearl barley.
Rice.

Soak 1 tablespoonful of the cereal overnight in 1½ pints of slightly salted water. Cook in the same water for 1½ to 3 hours in double cooker. If the mixture boils to less than 1 pint, add water to bring it up to that amount. When the flours are used,

one-half tablespoonful only should be used and the overnight soaking omitted. The flour should be made into a paste with cold water and added to the water in the double boiler.

CEREAL GRUELS

Proceed in the same way as for making cereal waters, but use 2 or 3 times the quantity of cereal called for.

CEREAL JELLIES

The best cereals to use are oatmeal, farina, rice, sago and rye. Take 2 level tablespoonfuls of cereal to 10 ounces of salted water. Boil 1 hour. Strain through cheese-cloth or hair seive. Turn into cups and put in a cool place. If it is desired to sweeten, 1 teaspoonful of sugar may be added and cooked into the mixture. The cereal may also be cooked in meat stock.

RICE IN MILK

Take 3 tablespoonfuls of rice to a quart of milk. Cook slowly in half the milk until the rice is twice its size, then add the rest of the milk. Sugar to taste. Add flavoring if desired. Cook slowly to a cream.

TOASTED BREAD CRUMBS

Place stale bread in a baking pan in the oven and let dry until brittle; roll with a rolling-pin into crumbs, after which they may again be placed in the oven and slightly browned. These may be used in puddings, with fruit sauces or juices, in soups or softened in milk and cooked with an egg.

FLOUR BALL (PARTIALLY DEXTRINIZED STARCH)

Make a cheese-cloth bag. Into the bag place 2 pounds of wheat flour. Put the bag into 2 quarts of cold water, let it come to a boil and boil for 5 hours. Remove the mass from the bag and place it in an oven and bake slowly for 3 hours. Break open the hard crust and grate or grind the softer inner core into a powder.

This is a very valuable form of carbohydrate especially for use in an infant suffering from proteolytic diarrhea. However, there are a number of proprietary foods on the market which are prac-

tically of the same composition. Imperial Granum, Dennos food, and Eskay's food are of this character. Mellin's food, Dextri-Maltose, and Allenbury's food are also mixtures containing dextrins in high proportion but in place of partially altered starch, they contain maltose as well as dextrin, a result of the fact that they are partially digested by the action of malt diastase.

SOUPS

MEAT STOCK

(Mutton, veal or beef may be used.)

Take 1 pound of meat, pass through a coarse grinder; add pinch of salt to 1 quart of cold water and add the meat. Allow to come slowly to a boil and simmer 3 hours, adding a little hot water from time to time. Cook down to 1 pint. Strain and put in a cool place until fat has risen and hardened. Remove the fat completely. This stock is the basis of making broths and soups.

CHICKEN STOCK

(The old fowls are better than the young for this purpose and they are less expensive. The type known in the markets as "fricaseé fowls" are best.)

Take 1½ to 2 pounds of fowl and chop into small pieces, cover with 2 or 3 pints of cold, salted water, bring to a boil and simmer over a slow fire for 3 or 4 hours. Add hot water from time to time. Let boil down to 1½ pints. Put in a cool place and allow fat to rise and harden. Remove fat completely. Strain and put in a cool place.

BEEF JUICE

Take ½ to 1 pound of round steak. Broil it slightly. Cut in two across the grain with a sharp knife. Squeeze out the juice with a meat press or potato ricer and add a pinch of salt. Feed fresh, or warm before serving, but do not heat sufficiently to coagulate the albumen.

(Beef juice has a low food value but it is valuable as an appetizer. The possibility of tape worm infection should always be kept in mind.)

MILK SOUP

Use one-third stock; two-thirds milk and salt to taste. Add level teaspoonful of butter. Bring to a boil. Pass through a fine sieve, two tablespoonfuls of either boiled potato, carrot, green peas, spinach, sorrel or root artichoke. Add to the mixture and boil for a few minutes. It is best to boil the potatoes with the skins on and peel them just before putting them through the sieve. All vegetables are to be thoroughly cooked. Dried vegetables are useful if they are well cooked and puréed.

VEGETABLE SOUPS

Add any of the vegetable purées, see below, to boiling stock.

CEREAL SOUPS

Add any of the cereal gruels or jellies, page 596, to boiling stock.

VEGETABLE-CEREAL SOUPS

Add any of the vegetable purées, see below, and cereal gruels, page 596, to boiling stock.

VEGETABLE PURÉES

Vegetables Available.—Spinach, celery, lettuce, carrots, asparagus tips, artichoke-heart or artichoke-root, endive, string beans, green or dried peas, dried beans or lentils, fresh, canned or dried corn. There are few vegetables, thoroughly cooked and properly puréed that are contraindicated in the diet of an infant during its second year. Even during its first year, small amounts of the more succulent vegetables, especially the iron containers, are useful additions to the diet.

Cooking.—All green vegetables (except asparagus) should be placed in *boiling* salted water and cooked for 30 minutes. Steaming is the preferred method of cooking the leaf vegetables. When this method is chosen, the vegetables should be thoroughly washed, soaked in cold salt water for half an hour and transferred to the upper section of a steam cooker. Dried vegetables should be soaked in cold water overnight before they are cooked. A little soda may be added to the water and they should be cooked for several hours.

Puréeing.—Pass the thoroughly cooked vegetables through a

puréeing sieve (Fig. 53) with a to and fro motion of the potato masher. For young babies, a fine sieve should be used. The resulting purée should be almost impalpable. For older infants, a coarser sieve may be used. Purées should be fed by spoon.

A meat grinder with a fine nut-butter attachment may be used in preparing spinach for young babies.

Puréeing of food should not be continued for too long a time; it makes the process of teaching the child to eat coarse vegetables too difficult later on.

MEAT-PULP

Take a piece of beef or mutton, sear on hot plate enough to attain the thermal death point of any contained parasite, split crosswise with the fiber with a sharp knife, scrape off the pulp with a dull knife or a spoon. For older infants, the meat may be cut in small pieces and passed through a meat grinder that has a nut-butter attachment. For very young infants who cannot take milk protein, the ground meat-pulp may be further comminuted with a mortar and pestle; or dried beef may be cut into small pieces and further dessicated in a slow oven. When dry, grind up in a coffee mill or a meat grinder.

MEAT-BALLS

Stir a beaten egg into meat-pulp to make a sticky mass. Drop small balls of the mass into boiling stock and cook for a few minutes.

MEAT-JELLY

Soak ½ box of shredded gelatin in cold water for 30 minutes. Add 2 cups of boiling stock and dissolve. Add 1 to 2 ounces of scraped or pulped meat. Cook for a few minutes over flame.

Some infants who will not take meat in other forms will accept it as a jelly.

DESSERTS

GELATIN-JELLY

Soak ½ box shredded gelatin in cold water for 30 minutes. Add two cups of boiling water and dissolve. Sweeten to taste. Add 1 ounce of lemon juice or 5 or 6 ounces of orange juice. Strain through fine sieve or cheese-cloth and set in a cool place.

GELATIN-MILK

Take 1 tablespoonful shredded gelatin, soak in 2 ounces cold water for 30 minutes, add boiling water to make 8 ounces and dissolve. With a teaspoonful of powdered arrowroot, make a paste with cold water. Add this paste to 4 ounces of top 16 ounce milk. Sweeten with a level teaspoonful of sugar. Boil over a flame with constant stirring for 10 minutes. Put in a cool place.

This is a mixture well tolerated by some infants who do badly on ordinary formulas.

FRUIT-GELATINS

Make a gelatin solution, see gelatin-jelly, and add any well-puréed fruit pulp. Chocolate may be used if desired.

FRUIT-PULPS

Available fruits:

Prunes, apricots, peaches, pears, apples, pineapple, nectarines; berries may be used unless they produce urticarial rashes in a given infant. Fruits may be either fresh, dried or canned. There is no objection to the use of canned fruits of standard brand such as the Del Monte. Dried fruits, while somewhat less palatable, have as much food value.

Cooking.—Fresh fruits should be peeled and cut into small bits. Berries should be thoroughly washed, put into cold water and cooked ½ to 1 hour. Pass through a puréeing sieve (Fig. 53) with to and fro motions of a wooden potato masher. For young infants a fine sieve should be used. The sieve should remove all seeds from berries. Sweeten to taste.

Dried fruits should be thoroughly washed through several waters. The less expensive, unbleached grades are preferable. Soak overnight in cold water and boil in same water until thoroughly cooked. Sweeten to taste. Pass through a puréeing sieve.

RAW FRUITS

Ripe apples, peaches, pears, bananas, alligator pears, (avacados), fresh prunes, sweet plums, and nectarines may be given to children after their first year. The avacado is rich in fat, and easily assimilated. It is necessary that fruits be given in finely divided particles. Scraping the fruit with the end of a spoon is a satisfactory method. Fruits should always be peeled, and

berries be seeded by sieve before being given to a child. Fruits with coarse pulps such as oranges and pineapples should be interdicted. The juice may be given, but the whole fruit, never.

FRUIT JUICES

The juice of oranges, grapefruits (pomelo), grapes, pineapples, loganberries, and raspberries may be given to infants at a very early age. Canned or bottled juices of standard brand are not contraindicated. The juice of stewed dried fruits is acceptable and is less expensive.

FRUIT JELLIES

Fruit jellies and marmalades without seeds are well tolerated by most infants. They may be added to bread crumbs, cereals and puddings and they meet the natural physiological demands for carbohydrate. Sugar given in this form is better tolerated than in the form of candy.

FRUIT DRINKS

For older infants, fruit juices diluted with water, make pleasant drinks, especially for use in hot weather. Water drinking may also be encouraged in the sick child in this manner. Reinforced with lactose, such drinks are also useful to supply energy needs for patients from whom it is desirable to withhold milk.

HIGH CALORIE ORANGE JUICE

Take 6 ounces of orange juice and add it to 12 ounces of water in which 8 tablespoonfuls of milk sugar have been dissolved by heat. Sweeten if necessary. Cool and give during the day.

Any fruit juice may be substituted for the orange juice. This mixture is usually eagerly taken and it provides about 200 calories.

FRUIT ICES

Any fruit juice diluted and sweetened may be frozen in an ice cream freezer. They are useful in treating fever patients.

BAKED BANANAS

Remove the skin, scrape off the outer pulp; place the fruit in a porcelain pan, sprinkle with sugar, cover and bake until

soft. Squeeze on a little lemon juice to flavor. Or, raise one section of the skin, add a small piece of butter and a little sugar, flavor with lemon juice and bake until soft.

BANANA FLIP

Scrape a peeled banana, pass through a sieve, add sugar and pineapple or orange juice to flavor. Beat the white of an egg to a stiff froth. Add the fruit pulp and beat together. The mixture may be frozen if desired.

APRICOT JELLY

Soak 1 pound of dried apricots overnight; cook until soft. Dissolve ½ box of shredded gelatin in cold water. Add 4 tablespoonfuls of sugar. Rub the apricots through a fine sieve, add to the gelatin. Fold in the stiffly beaten whites of 4 eggs; pour into moulds. When set, serve plain or with milk.

Any fruit pulp may be used instead of the apricot if desired.

FRUIT SYRUP (FOR CONSTIPATION)

Fill a fruit jar with washed, dried apricots. Steam. Pour in brown corn syrup to completely fill the jar. Cover tightly. Stand in a pan of water in a slow oven until all the syrup is absorbed by the fruit. Serve as a preserve at one or two meals a day. If this fails to be laxative enough, at the next making add a dozen or more senna leaves to the mixture.

PUDDINGS

EGG-APPLE-SAUCE DESSERT

Into three tablespoonfuls of very hot apple sauce, beat the yolk of an egg; spread the separately beaten whites over the top and set in the oven to allow the egg to set. Any sort of fruit sauce may be used in place of apple if desired.

ROMAN CREAM

Take 1 pint of milk, one-tenth box of gelatin, yolks of 2 eggs, 3 tablespoonfuls of sugar, vanilla to taste. Dissolve the gelatin in the milk and place in a double cooker. Beat the eggs and sugar together, add to the milk mixture and cook until it thickens.

being careful not to boil too long. Take off the fire, and stir in the beaten whites of the eggs. Cool and serve plain or with milk.

BROWN BETTY

Cover the bottom of a baking dish with toasted bread crumbs. On top of them, place a layer of sliced apples or bananas. Sprinkle with sugar and lay on bits of butter. Repeat the process until the dish is full. Moisten with a little water and lemon juice. Cover and bake for ½ hour. Remove cover, and bake for ½ hour more.

STEAM PUDDING

Take ½ large cup of sugar, ½ teaspoonful salt, 1 whole egg, 1 tablespoonful melted butter and beat them all together. Then add 1 cup of milk and two small cups of flour sifted in gradually. Two level teaspoonfuls of baking powder should be added to the second cup of flour. Butter a mould and steam 2 hours or more. Serve with a fruit sauce.

CORNSTARCH PUDDING

Mix into a pint of milk 1 teaspoonful butter, 3 teaspoonfuls sugar, a pinch of salt. Two tablespoonfuls of cornstarch should first be made into a paste with a little of the milk. Then bring the milk mixture to a boil and pour it over the starch paste, stirring constantly. Boil for 10 or 15 minutes.

ORANGE STARCH PUDDING

Make a cornstarch paste with cold water. Boil orange juice, (dilute if desired) and stir in the starch paste, stirring constantly. Boil 10 or 15 minutes.

APPLE SNOW

Take 1 cupful of hot apple sauce pulped; beat the whites of 2 eggs stiff, add 1 tablespoonful of sugar and stir into the apple sauce; set aside to cool. Any cooked fruit pulp may be used.

RICE, SAGO OR TAPIOCA PUDDING

Prepare a custard. See page 596. If rice is used, it should be well cooked; if tapioca or sago, it should have been soaked over-

night. Mix the cereal with the custard, set in a pan of water in a moderate oven. Cook the tapioca mixture 30 to 45 minutes; the rice or sago mixture 10 to 15 minutes.

FROZEN DESSERTS

VANILLA ICE CREAM

Scald 1 pint of milk, add 3 well beaten eggs and cook in a double boiler until it is as thick as boiled custard. Remove from the fire, add 1 cup of sugar and when cold, 2 teaspoonfuls of vanilla, ⅓ level teaspoonful of salt and 2 cups of thin cream. Freeze, and set aside to ripen before serving.

FRUIT ICE CREAM

Fruit pulps may be added to the cream when it is half frozen. Finish the freezing and pack down.

BREAD CRUMBS ICE CREAM

Add one cup of toasted bread crumbs to the cream mixture before freezing.

FRUIT SHERBET

Boil 2 cups of sugar in 1 pint of water to form a syrup. Add 1 pint of water and 1 pint of fruit juice and the juice of 1 lemon. Mix thoroughly and freeze.

CHAPTER XXIV

DRUGS

During the last generation the use of drugs in the treatment of disease fell into unwarranted disfavor, very largely because medical men of the previous generation had held too great a faith in them and had overused them with inexact conceptions of what results ought to be expected. There followed a reaction in which medical teaching swung to the opposite extreme, and therapeutic nihilism became almost a fashion. But young physicians, thrown upon their own resources, soon learned that the demand for therapeutic measures was exacting and inevitable if they were to successfully cope with disease. At this time, scores of manufacturing pharmacists further complicated the situation by attempting to meet the needs of the physician by providing him with a host of remedies, fantastically named and with a therapeutic literature, extensive and wonderful in its misinformation.

Fortunately today, the study of pharmacology and of rational therapeutics is coming into its own, and more and more, men are accepting drugs as they should be accepted, considering them only as adjuvants to a therapeutic regime and using such of them as have been proved by experience to be of value.

There is still too much unwarranted criticism from men who forget that medicine is not yet an exact science and that it cannot be such until our knowledge of the fundamental branches, especially physiology, becomes more exact. To entirely abandon the clinical observations of thousands of thoughtful observers of the last three generations, because we are unable to bring them into accord with facts deduced from animal experimentation, is certainly not wise. In this connection, it is interesting to remember that the successful treatment of syphilis and of malaria for years was based on empiricism; it is only in recent time that experimenters have demonstrated the rationale of such therapeusis. It is not improbable that many of the unexplained results that we now obtain by the empirical use of drugs in other diseases, may one day be justified by the results of experiment. At any event, the body of medical experience warrants the continuance of the use of certain well-tried drugs in the treatment of disease,

even if we are without a satisfactory explanation of their apparent effectiveness.

The secret of the successful use of drugs depends on the knowledge of their physiologic action; on the use of adequate dosage; and on a recognition of the full therapeutic effect. Once the desired effect is attained, the drug should be discontinued. Complex formulas are to be deprecated although a well-written prescription in which a number of ingredients are combined to reinforce one another, should not be stigmatized as reprehensible.

The drugs used, particularly in the practice of pediatrics, need not be many. The writers recognize that different drugs have similar actions and that various clinicians have favorite formulas, any of which may be used with advantage. No attempt is made in this chapter to deal with or to mention every drug that may be valuable in the treatment of sick infants. The writers desire to deal only with those drugs and formulas which they have found, in actual practice to be of value to them. Failure to mention a therapeutic agent does not mean that it is condemned. Undoubtedly, the ability to use a few drugs with which he is thoroughly familiar, is of much greater advantage to the practicing physician than to possess slight knowledge of a long list of remedies.

There are few peculiarities of childhood that influence the action of drugs. Much has been written about the tolerance of infants for atropin. It is true that there are a few infants who are exceedingly resistant to the drug; but it is also true that there are others who are equally susceptible to its effect. The idiosyncrasies to opium among infants, are equally striking. It is wise, therefore, when dealing for the first time with a child for whom it is found necessary to prescribe opium, to begin with a small dose in order to determine the tolerance of that individual for the drug. A helpful practice in prescribing opiates is not to add them to mixtures but to give them alone, in order that the dose may be readily controlled.

A convenient method of computing the dose of a drug to be given an infant is to use its weight as an index. Assuming the usual adult to weigh 150 pounds, it is a simple matter to determine the dose of the drug to be given the baby, by using the child's weight as a numerator and the average adult weight as a denominator; the fraction thus obtained is the fraction of the average adult dose. In using opium, however, for the first time,

it is better to halve the dose as estimated in this way, and to gradually increase it as the infant's tolerance is determined.

In the following prescriptions, *the dosage given is that appropriate for a child approximately one year old who weighs about 22 pounds.* A three months child may be given one-third dose; a six months child, one-half; and a nine months child, two-thirds. The prescriptions are written for a single dose only, as it is probable physicians will desire to vary the total amount of the drug prescribed to meet the needs of individuals.

ANEMIA

Mild secondary anemia:

℞ Mercury and chalk powder, gr. 1/3
Saccharated carbonate of iron, gr. 2.
S.: Give one such powder moistened with milk after each meal.

Anemia accompanied by malnutrition:

℞ Syrup iodid of iron, m. 5
Emulsion cod-liver oil (33%)*, dr. 3/4
Simple elixir to flavor.
S.: Give one teaspoonful before each meal.

℞ Compound elixir glycerophosphate, with strychnine, m. 30
S.: Give one-half teaspoonful before each meal.

Anemia accompanied by anorexia:

℞ Compound elixir glycerophosphate, with strychnine, m. 30
Emulsion cod-liver oil (33%), dr. 1
Simple elixir to flavor, m. 30
S.: Two teaspoonfuls three times a day.

In the more profound secondary anemias or in the moderate anemias with marked disturbances of nutrition, hypodermic injections of iron cacodylate are of advantage. Five to 8 minims of a 5 per cent solution in sterile ampoules, are to be injected into the triceps muscles daily for 7 to 10 days. The same dose may be injected every other day for a like number of days.

The organic iron found in spinach is very effective as a blood regenerator; it is readily obtainable at all seasons, (the canned product is acceptable), and it may be given to babies as young as three months. It should be finely puréed and may be given in doses of one teaspoonful to one tablespoonful daily. Scraped beef and pulped liver are indicated additions to the diet.

*The writers prefer 33 per cent emulsion; however, there is no objection to the 50 per cent product often employed.

ASTHMA

At the time of the onset of the attack:

℞ Atropin sulphate, gr. 1/500
Adrenalin, (1 to 3500), m. 8
S.: Inject into the triceps muscle*.

℞ Atropin sulphate, gr. 1/400
Adrenalin (1 to 1000), m. 10
S.: One drop under the tongue every minute for 5 minutes. Repeat in 4 hours if necessary and again in 8 hours.

During attack:

℞ Spirits of chloroform, m. 3
Terebene (emulsify together with tragacanth), m. 2
Bromide of soda, gr. 5
Simple syrup
Distilled water of each to make dr. 1
S.: One teaspoonful every 3 or 4 hours.

As a spray:

℞ Dionin, 2%
S.: To be used as a spray by physician or nurse only, every 8 or 10 hours for 48 hours.

Following the acute attack:

℞ Fowler's solution, m. 2
Syrup iodid of iron, m. 15
Emulsion cod-liver oil (33%), to make dr. 2
S.: Two teaspoonfuls after each meal.

BITES (INSECT)

℞ Aluminum acetate, gr. 3
Phenol, gr. 3
Glycerin, dr. 2
Water to make oz. 1
S.: Apply locally.

If infected with pyogenic organisms:

℞ Thymol di-iodid, dr. 1
Zinc stearate, oz. 1
S.: Use as a dusting powder.

(NOTE: On rare occasions, this injection may be followed by pallor and dyspnea. These symptoms pass off, but the use of 1/2 grain caffein sodium benzoate by hypodermic will dissipate them promptly.)

BRONCHITIS AND BRONCHOPNEUMONIA

Mustard pack and applications; drugs are adjuvant.

During acute attack with bronchial spasm:

℞ Spirits of chloroform, m. 3
Terebene (emulsify together with tragacanth) m. 2
Bromide of soda, gr. 5
Simple syrup,
Distilled water of each to make dr. 1
S.: One teaspoonful every 3 or 4 hours.

or

℞ Syrup hydriodic acid, m. 20
Spirits chloroform, m. 2
Syrup raspberry, dr. 1/2
Water, to make dr. 1
S.: Give 1 teaspoonful every 3 hours until secretions are free.

To allay night cough and secure rest:

℞ Codein, gr. 1/20
Bromide of soda, gr. 3
Syrup raspberry, to make dr. 1
S.: Give 1 teaspoonful at 6 P.M. May repeat *once* after 4 hours if child is awake and coughing.

In severe bronchitis or bronchopneumonia with cyanosis:

℞ Atropin sulphate gr. 1/600
S.: Inject into triceps muscle. Repeat in 4 to 6 hours if necessary.

℞ Camphor (10%) in olive oil, (sterile in ampoules.)
S.: Inject 10 or 12 minims into triceps every 2 to 4 hours as indicated.

To promote secretion:

The ammonium salts are often used as expectorants and they are undoubtedly of value but the writers' experience leads them to prefer the iodides.

℞ Ammonium chloride, gr. 3
Glycerin, m. 10
Syrup raspberry, dr. 1/2
Water, to make dr. 1
S.: Give 1 teaspoonful every 3 hours.

With laryngeal and tracheal involvement:

℞ Ipecac, gr. 1/100
Antimony and potassium tartrate gr. 1/100
Make into tablet triturate.
S.: Give 1 tablet in sweetened water every 30 minutes until 10 have been given.

To stimulate secretion with moisture:

℞ Compound tincture benzoin, dr. 1
Oil of pine (or Oil of eucalyptus) dr. 1/2
S.: Use in steam inhalations.

After acute stage as reconstructive measure:

℞ Syrup iodid of iron, m. 20
Emulsion cod-liver oil (33%), dr. 1
Simple elixir to flavor.
S.: Give 1 teaspoonful every 4 hours.

For chronic persistent cough:

℞ Creosotal, m. 2
Emulsion cod-liver oil (33%), dr. 1
Simple elixir to flavor.
S.: Give 1 teaspoonful every 4 hours.

To support heart in severe cases of bronchopneumonia:

℞ Strophanthin, gr. 1/1500
S.: Inject into a vein in emergency.

℞ Tincture digitalis
S.: Give 6 minims in water by mouth every 4 hours until pulse slows; then reduce dose to 1 to 2 minims.

BURNS

℞ Bismuth subcarbonate, dr. 4
Castor oil, oz. 1
S.: Apply to surface.

℞ Sodium bicarbonate, dr. 2
Water, oz. 16
S.: Wring out compresses and apply as emergency measure.

℞ Zinc oxide ointment,
Petrolatum, of each oz. 1/2
S.: Spread on lint and apply to parts.

CHICKEN-POX

℞ Cresol, dr. 2
Water, gallon 1
S.: Bathe child daily to prevent itching and secondary infection.

℞ Thymol di-iodid, dr. 1
Calamine lotion, oz. 6
S.: Shake well and apply.

℞ Calamine, gr. 40
Zinc oxide, dr. 1
Liquor aluminum acetate, dr. 1
Phenol, m. 1
Glycerin, dr. 1
Rose water, to make oz. 1
S.: Apply locally.

(NOTE: This is known as calamine lotion.)

If lesions become impetiginous:

℞ Thymol di-iodid, dr. 2
Zinc stearate, oz. 1
S.: Dust parts after removing crusts with green soap compresses.

℞ Ammoniated mercury powder, g. xx
Calamine lotion oz. 3

COLIC

For mild intestinal colic:

℞ Cinnamon water
S.: Add 1 teaspoonful to each feeding.

For moderately severe cases of colic:

℞ Atropin sulphate, gr. 1/1200
Spirits chloroform, m. 1
Sodium bicarbonate, gr. 5
Simple elixir, dr. 1
S.: Give 1 teaspoonful every 2 hours for 3 or 4 doses.

℞ Spirits nitrous ether
S.: Give 30 drops in water every 1/2 hour for 3 or 4 doses.

For severe colic with distention and respiratory embarrassment:

℞ Sodium bicarbonate, oz. 1
Water at 110° F., qt. 1
S.: Use for colonic flushing.

℞ Atropin sulphate, gr. 1/800
Antipyrin, gr. 1/2
Caffein soda benzoate, gr. 1/8
Powdered calcium carbonate, gr. 5
Saccharin, gr. 1/12
Make into one powder.
S.: Give 1 powder every 2 hours for 3 or 4 doses.

℞ Milk of magnesia
S.: Give 3 teaspoonfuls and repeat in 12 hours if necessary.

(NOTE: Milk of magnesia is often given in too frequent and too small doses and much intestinal colic is due to this cause.)

℞ Pituitrin, m. 4
S.: Inject into triceps muscle. Useful when abdominal distention is extreme.)

COLITIS (MUCOUS)

As adjuvants to hygienic and dietetic measures:

℞ Atropin sulphate, gr. 1/1200
Powdered carbonate of iron (saccharated) gr. 2
Mercury with chalk, gr. 1/2
Powdered calcium carbonate, gr. 5
Make into one powder.
S.: Give 1 powder every 3 hours.

℞ Aluminum acetate, dr. 2
Water, 110° F., qt. 1
S.: Use for colonic flushing.

For acute colitis (noninfectious):

℞ Castor oil, m. 10
Paraffin oil to make, dr. 2
Acacia to emulsify
S.: Give 2 teaspoonfuls every 4 hours for 3 or 4 days.

CRETINISM

℞ Thyroid extract, gr. 1/4
S.: Give 1 to 4 tablets in ascending dosage, 3 times a day according to conditions.

CONSTIPATION

℞ Malt-soup extract, oz. 16
S.: For bottle fed infant give 1/4 to 1/2 ounce daily, divided equally into feedings; for breast nursling, same dosage dissolved in 6 ounces of water and 1/2 to 1 ounce to be given before each nursing.

℞ Lactose, oz. 1-1/2
Hot water, (boiling), oz. 3
Orange-juice, oz. 5
S.: Give 8 ounces during 24 hours.

℞ Lactose oz. 1-1/2
S.: Mix with food during 24 hours.

(NOTE: For older infants only.

℞ Milk of magnesia,
S.: Give 2 to 3 teaspoonfuls at bedtime, repeated in the morning if necessary.

℞ Sodium sulphate, gr. 6
Cinnamon water, dr. 1
S.: Add 1 teaspoonful to each feeding.

For chronic constipation adjuvant to hygienic and dietetic measures:

℞ Paraffin oil, dr. 1
Acacia to emulsify,
S.: Give 1 teaspoonful 3 times a day.

℞ Atropin sulphate, gr. 1/1600
Fluid extract cascara, aromatic, m. 20
Sodium bicarbonate, gr. 5
Tincture nux vomica, m. 1
Elixir pancreatin, dr. 1
S.: Give 1 teaspoonful 3 times a day after meals. Gradually decrease dose until medicine is withdrawn.

To produce immediate evacuation:

℞ Paraffin oil, oz. 2
S.: Inject warm into rectum.

℞ Sodium bicarbonate, oz. 1
Water, warm, pt. 1
S.: Inject slowly into rectum.

℞ Gluten suppositories or soap sticks,
S.: Insert suppository into rectum.

(NOTE: Glycerin suppositories are always irritating. Soap sticks may become so with too frequent use).

CONVULSIONS DURING PAROXYSMS

Mustard pack. (See Methods).

Chloroform inhalations sufficient to obtain relaxation.

℞ Morphin sulphate, gr. 1/40
or
Codein phosphate, gr. 1/20
S.: Inject hypodermically.

Between the attacks:

℞ Chloral hydrate, gr. 3
Sodium bromide, gr. 5
Distilled water, dr. 1
S.: Give 1 teaspoonful every 3 hours for 3 or 4 doses. Double the dose if given by rectum.

℞ Sodium bicarbonate, oz. 1/2
Water, 110° F., pt. 1
S.: Inject slowly into rectum.

CORYZA

℞ Atropin sulphate, gr. 1/1600
Spirits camphor, m. 1
Sodium bromide, gr. 1/2
Syrup raspberry,
Water, of each m. 15
S.: Give 15 drops every hour for 6 doses, then every 3 hours for a like period.

℞ Borax, dr. 1/2
Water, 100° F., oz. 6
S.: For nasal irrigation.

℞ Gray oil, m. 20
Adrenalin ointment, gr. 20
Light petroleum oil to make dr. 11
S.: Instil small amount in nares every 4 hours.

℞ Argyrol, dr. 1
Distilled water, oz. 1
S.: 10 drops in nostrils 3 times a day.

COUGH

For pharyngeal cough:

℞ Silver nitrate, dr. 1
Distilled water, oz. 1
S.: Apply locally to tonsils and fauces not oftener than once a day.

℞ Syrup iodid of iron, m. 10
Syrup of raspberry,
Water, of each, m. 10
S.: Give one-half teaspoonful every 2 or 3 hours.

As demulcents:

Marshmallow.
Petrolatum emulsion.
Honey and milk.
Flaxseed lemonade.

For laryngeal cough:

℞ Ipecac, gr. 1/100
Antimony and potassium tartrate gr. 1/100
Make into tablet triturate.
S.: Give 1 tablet dissolved in sweetened water every half-hour until 10 or 12 are taken.

℞ Oil of pine, dr. 1
or
Oil eucalyptus, dr. 1
or
Compound tincture benzoin, dr. 1
Water, pt. 2
S.: Use as steam inhalations.

(NOTE: Plain steam is usually just as effective.)

℞ Codein, gr. 1/20
S.: Give 1 tablet dissolved in sweetened water. Repeat once after 4 hours if necessary.

For bronchial cough:

Steam inhalations. (Add medicaments if desired.)

℞ Sodium iodid, gr. 3
Spirits chloroform, m. 2
Syrup raspberry,
Water, of each to make, dr. 1
S.: Give 1 teaspoonful every 3 hours during early stages.

During stage of irritation without secretion:

℞ Ipecac,
Antimony and potassium tartrate, of each, gr. 1/100
Make into tablet triturate.
S.: Give 1 tablet every one-half hour until 10 have been taken.

When secretion is established:

℞ Creosote carbonate, m. 1
Syrup of chocolate, dr. 1
Emulsify with acacia.
S.: Give 1 teaspoonful every 4 hours.

During the stage of defervescence:

℞ Syrup iodid of iron, m. 20
Bromide of soda, gr. 2
Emulsion cod-liver oil (33%), dr. 2
Flavor with simple elixir.
S.: Give 2 teaspoonfuls 3 times a day after meals.

NOTE: The slight liberation of iodin is unimportant.)

For the constant irritative cough of bronchopneumonia:
Mustard pack.

℞ Sodium iodid, gr. 3
Spirits chloroform, m. 2
Syrup raspberry,
Water, of each to make dr. 1
S.: Give 1 teaspoonful every 3 hours.

For irritative cough of pleurisy accompanying lobar pneumonia:
Mustard plaster applied locally.

℞ Codein, gr. 1/20
S.: Inject hypodermically; repeat once after 4 hours if necessary.

For cough of pertussis:

℞ Antipyrin, gr. 1
Sodium bromide, gr. 2
Caffein sodium benzoate, gr. 1/4
Glycerin,
Water, of each to make dr. 1
S.: Give 1 teaspoonful every 4 hours.

During stage of defervescence of pertussis:

℞ Quinine ethyl-carbonate, gr. 1
Chocolate syrup, dr. 1
Emulsify with acacia.
S.: Give 1 teaspoonful every 4 hours.

During convalescence:

℞ Syrup iodid of iron, m. 10
Emulsion cod-liver oil (33%), dr. 2
Flavor with simple elixir.
S.: Give 2 teaspoonfuls every 4 hours.

CROUP

(NOTE: A throat culture is essential to exclude laryngeal diphtheria).

℞ Ipecac,
Antimony and potassium tartrate,
of each, gr. 1/100
Make into tablet triturate.
S.: Give 1 tablet dissolved in sweetened water every 15 minutes until 10 are taken.

℞ Iodized calcium, gr. 1/3
S.: Give 2 tablets in sweetened water every half-hour until 20 are taken.

As an emergency measure:

℞ Apomorphin, gr. 1/100
S.: Inject hypodermically.

CYANOSIS

Mustard pack.

Oxygen (see page 557).

If respiratory in origin:

℞ Atropin sulphate, gr. 1/800
S.: Inject hypodermically.

If cardiac in origin:

℞ Strophanthin, gr. 1/1500
S.: Inject intravenously or intramuscularly as emergency measure.

℞ Camphor (10%), in olive oil, sterile ampoules.
S.: Inject 10 minims hypodermically.

℞ Tincture digitalis,
S.: Give 5 minims every 4 hours until pulse slows, then reduce the dose to 2 minims.

If bowels are distended:

℞ Pituitrin, (surgical), m. 4
S.: Inject hypodermically.

CYSTITIS

℞ Potassium citrate, gr. 8
Syrup citric acid, m. 10
Water, to make, dr. 2
S.: Give 2 teaspoonfuls every 2 hours followed by copious draughts of water, *day and night* until the urine reaction is alkaline.

℞ Hexamethylenamine, gr. 40
S.: Dissolve in 1 pint water and use as beverage or in preparation of formulas during 24 hours.

(NOTE: Be certain urine is acid when using this drug.)

To render urine acid:

℞ Acid sodium phosphate, gr. 5
Syrup of orange, dr. 2
S.: Give 2 teaspoonfuls in water every 2 or 3 hours until urine reaction is acid.

For chronic forms not responsive to above:

℞ Sodium benzoate, gr. 2
Salol, gr. 3
Sugar, gr. 15
Make into one powder.
S.: Give 1 powder in a little fruit jelly every 3 hours.

Irrigate bladder with normal saline solution.

℞ Argyrol, or mercurochrome, gr. 10
Distilled water, dr. 1
S.: Instil into bladder after lavage.

DIARRHEA

(NOTE: *All medicines are adjuvant to dietetic and hygienic measures*).

Acute diarrhea at beginning of attack:

℞ Castor oil, dr. 2
S.: Give from warm spoon.

To restore blood volume:

℞ Normal salt solution (0.8%), oz. 4 to 8
S.: Inject into peritoneum.

℞ Glucose, 10 per cent in normal salt solution oz. 2 to 4
S.: Inject intravenously.

℞ Acacia, 7 per cent,
Glucose, 5 per cent,
Normal salt solution, oz. 2 to 4
S.: Inject intravenously.

To meet toxemia of dysenteric diarrhea:

℞ Antidysentery serum 30 c.c.
Normal salt solution 500 c.c.
S.: Inject intravenously. Repeat at 8-hour intervals till symptoms abate.

To inhibit excessive peristalsis:

℞ Morphin sulphate, gr. 1/50
S.: Inject hypodermically; repeat after 4 hours if needed.

℞ Deodorized tincture opium, m. 1/2
S.: Give 1/2 minims in water every 4 hours for 3 or 4 doses.

(NOTE: Opium given with bismuth or the other astringents is often effective but the good result is probably due to the narcotic rather than to the astringent.)

For colitis and tenesmus with diarrhea:

℞ Sodium bicarbonate, oz. 1/2
Water, pt. 1
S.: Flush bowels.

℞ Aluminum acetate, dr. 1
Water, pt. 1
S.: Use for bowel flushing if mucus and blood be present.

℞ Starch, dr. 2
Water, pt. 1
S.: Use as enema for protective action of starch.

(NOTE: Opium added to starch enemas is of little or no effect.)

For collapse in diarrhea:

Mustard pack. (See Methods.)

For distention:

℞ Pituitrin, (surgical), m. 4
S.: Inject hypodermically; may repeat in 2 hours if needed.

For subacute diarrheas:

℞ Castor oil, m. 5
Liquid petrolatum, white, m. 25
Acacia to emulsify.
S.: Give one-half teaspoonful every 2 or 3 hours.

℞ Deodorized tincture of opium, m. 1/2
S.: ½ minims in water may be given if irritation becomes great.

℞ Compound chalk mixture, dr. 1/2
S.: Give one-half teaspoonful every 3 hours.

For chronic diarrhea:

℞ Castor oil, dr. 2
S.: Give 2 teaspoonfuls at intervals of 1 week.

℞ Atropin sulphate, gr. 1/1600
Taka-diastase, gr. 2
Calcium carbonate, gr. 5
Animal charcoal, gr. 5
Saccharin, gr. 1/12
S.: Give 1 powder 4 times a day one-half hour after meals.

NOTE: Colonic flushings with 5% sodium bicarbonate solution are effective in the chronic type of diarrhea as well as in the acute type. If much mucus or blood be present, ¼% to 1% aluminum acetate solution may be substituted. Silver nitrate in 1/2% solution followed by normal saline solution may be used with advantage in many cases where ulceration is present. The complete evacuation of the silver solution must be assured. If blood persists, have the patient's serum agglutinated against cultures of dysentery bacilli.

DIPHTHERIA

Antitoxin, 10,000 units, (never less) should be injected intravenously or intramuscularly at once the diagnosis is made. The dose may be repeated in 12 to 24 hours.

For laryngeal type, (in addition to antitoxin):

℞ Calomel, gr. 20
S.: Place on hot metal plate and allow patient to inhale fumes under a tent once; may repeat on second and third day if necessary.

EARACHE

℞ Phenol, m. 1
Glycerin, dr. 1
S.: Instil 1 or 2 drops into ear.

℞ Cocaine, gr. 1
Adrenalin (1 to 1000), dr. 2
S.: Instil 1 or 2 drops into ear.

ECZEMA

For moist weeping facial eczema in infants:

℞ Black wash,
Milk of magnesia,
Rose water, of each, oz. 1
S.: Until oozing ceases, apply on compresses which are to be kept moist. Apply mask over compresses.

℞ Phenol, gr. 5
or oil cade, m. ii
Resorcin, gr. 10
Glycerin, m. 25
Lassar's paste, no acid,
Cold cream, U.S.P., of each to make, oz. 1
S.: Following previous prescription, when oozing ceases, apply locally under a mask.

When dermatitis is extensive:

℞ Ichthyol, dr. 1
Water, oz. 1
S.: Apply on compresses; change every half hour for 5 to 10 hours.

For the less acute stages:

℞ Coal tar solution, (N. F.) m. 20
Resorcin, gr. 10
Glycerin, m. 25
Lassar's paste, no acid,
Cold cream, U.S.P., of each to make, oz. 1
S.: Apply locally under a mask.

For indolent eczema and itching:

℞ Crude coal tar gr. 40
Zinc carbonate, gr. 20
Lassar's paste, with acid
Lanolin, of each to make, oz. 1
Misc.: Rub first two drugs together and add to paste and lanolin.
S.: Apply locally under mask.

For florid stage or when impetigo complicates:

℞ Ammoniated mercury, gr. 10
Phenol, gr. 5
Lanolin,
Cold cream, U.S.P., of each to make, oz. 1
S.: Apply locally under a mask.

To remove crusts:

℞ Green soap, dr. 2
Water, oz. 4
S.: Apply on compresses for 3 or 4 hours.

(NOTE: Washing the parts with tar soap solution is often sufficient.)

℞ Crude coal tar gr. 15
Zinc carbonate (rub well together) gr. 20
Lassar's paste, no acid,
Cold cream, U.S.P., of each to make, oz. 1
S.: Apply locally (with or without mask) after crusts have been removed.

For cleansing the skin:

℞ Liquid petrolatum, light
S.: Cleanse skin gently with cotton or fine gauze dabs.

For chronic indolent eczema with itching (seborrheic):

℞ Crude coal tar,
Ichthyol,
Salicylic acid aa, gr. 15
Acetone, q. s., oz. 1/2
S.: Apply lightly with brush to indolent patches until they are irritated.

For stage of defervescence:

℞ Zinc oxide, gr. 30
or
Calamine, gr. 30
Cold cream, U.S.P., to make oz. 1
S.: Apply locally under a mask.

For accompanying pityriasis capitis:

℞ Resorcin, gr. 30
Glycerin, m. 25
Precipitated sulphur, gr. 30
Petrolatum to make oz. 1
S.: To be applied at night, followed in the morning by tar soap shampoo.

As metabolic stimulants:

℞ Cinchophen, gr. 2
S.: Give 1 powder 3 times a day.

℞ Thyroid extract, gr. 1/10 to 1/4
S.: Give 1 tablet morning and evening.

℞ Fowler's solution
S.: Give 1 drop in water 3 times a day.

As a tonic:

℞ Syrup iodid of iron, m. 5
Emulsion cod-liver oil (33%), dr. 1
Flavor with simple elixir.
S.: Give 1 teaspoonful before each meal.

ENURESIS

As adjuvant to training of nervous system:

℞ Atropin sulphate, gr. 1/600
S.: Give a dose at 4 and 6 P.M.

EPILEPSY

As adjuvant to hygienic measures:

℞ Luminal, gr. 1/4
Powdered Sugar, gr. 5
S.: One powder at 6 P.M.

℞ Bromide of soda, gr. 30 to 50
S.: Replace sodium chloride in diet with this daily dose.

EPISTAXIS

℞ Solution Adrenalin, 1 to 3500
S.: Apply on cotton pledget.

EXCORIATIONS (OF BUTTOCKS)

For oozing type, as adjuvant to open treatment:

℞ Black wash,
Milk of magnesia,
Rose water, of each, oz. 1
S.: Apply on compresses frequently changed.

For dry type, adjuvant to open treatment:

℞ Thymol di-iodid, dr. 2
Zinc stearate, oz. 1
S.: Dust on parts.

℞ Zinc stearate, dr. 2
Castor oil, oz. 1
S.: Apply to parts.

As a preventive, adjuvant to dry diapers:

℞ Fuller's earth.
S.: Dust on buttocks.

℞ Lanolin
S.: Apply to parts.

FISSURES

Fissure in ano:

℞ Ichthyol, gr. 10
Belladonna ointment, gr. 30
Diachylon ointment, dr. 2
Lanolin to make, oz. 1
S.: Apply night and morning after mild digital dilatation of sphincter.

℞ Silver nitrate, 5% solution
S.: Apply locally to raw surface.

To maintain soft evacuations:

℞ Petroleum oil
Acacia to emulsify
S.: Give 1 teaspoonful 3 or 4 times a day.

Lip fissures:

℞ Camphor-phenol
Cold cream, U.S.P., of each to make oz. 1/2
S.: Apply locally.

Deep syphilitic fissures of lips:

℞ Mercury nitrate, gr. 1/24
Compound tincture benzoin, oz. 1
S.: Touch fissures daily.

FURUNCLES

Furuncles in infants:

℞ Black wash,
Milk of magnesia,
Rose water, of each, oz. 1
S.: Apply on compresses and keep moist.

℞ Green soap, dr. 1
Water, oz. 1
S.: Apply on compresses until after pus is evacuated.

GONORRHEA

For acute cases, girl infants:

℞ Normal saline solution, (0.8%)
S.: Irrigate vagina through speculum.

For subacute stage:

℞ Mercurochrome, 2% in water.
S.: Inject twice daily.
Or following Julius H. Hess.

℞ Silver nitrate, gr. 1
Lanolin,
Vaseline, of each to make oz. 1
S.: Fill vagina from catheter tipped glass syringe. Continue instillations for 1 week. Then replace silver nitrate in ointment by mercurochrome, 1%.

HEART DISEASE

For acute cardiac failure:

℞ Strophanthin, gr. 1/1500
S.: Inject intravenously or hypodermically.

℞ Camphor, (10%) in olive oil, sterile ampoules
S.: Inject 10 minims hypodermically.

Mustard pack.

For acute endocarditis:

℞ Methyl salicylate
S.: Give 3 to 5 drops in a little sugar every 4 hours.

℞ Sodium salicylate, gr. 30
Sugar, dr. 1
Water, 100° F. oz. 1
S.: Inject into rectum for retention, once daily.

For cardiac decompensation:

℞ Tincture digitalis
S.: Give 2 minims every 3 to 4 hours.

As a tonic:

℞ Fowler's solution m. 1
Phosphorus gr. 1/800
Emulsion cod-liver oil, (33%) dr. 1
Flavor with simple elixir.
S.: Give 1 teaspoonful 3 times a day.

IMPETIGO

For mild cases without crusting:

℞ Thymol di-iodid, dr. 2
or
Zinc stearate, oz. 1
S.: Use as dusting powder.

For oozing type:

℞ Black wash,
Milk of magnesia,
Rose water, of each, oz. 1
S.: Apply on compresses and keep moist.

℞ Thymol di-iodid, dr. 1
or
Ammoniated mercury, gr. 30
Phenol, m. 30
Zinc oxide, dr. 1
Calamine lotion, to make, oz. 3
S.: Apply locally.

To remove crusts:

℞ Green soap, dr. 1
Water, oz. 1
S.: Apply overnight, on compresses.

For ordinary crusty type:

℞ Phenol, m. 10
Ammoniated mercury, gr. 20
Lanolin,
Cold cream, U.S.P., of each to make oz. 1
S.: Apply locally.

ICHTHYOSIS

℞ Paraffin oil, light
S.: Apply to skin twice a day with gentle friction.

℞ Thyroid extract, gr. 1/10 to 1/4
S.: Give 1 tablet 3 times a day.

MALARIA

℞ Quinine hydrochloride, gr. 1
S.: Inject hypodermically, anticipating the chill if possible.

(NOTE: It is better to give a few large doses at the proper times than to use frequently repeated small doses).

℞ Quinine sulphate, gr. 2
Syrup of chocolate, dr. 1
Acacia to emulsify
S.: Give 1 teaspoonful twice a day until effective.

(NOTE: Hypodermic injections of quinine are often required when the drug given by mouth is not effective.)

MEASLES

For comfort of the skin:

℞ Compound solution of cresol, dr. 2
Water, gallon 10
S.: Give daily bath in this solution.

For the cough:

℞ Syrup hydriodic acid, m. 15
Spirits chloroform, m. 2
Bromide of soda, gr. 2
Syrup of raspberry,
Water, of each to make, dr. 1
S.: Give 1 teaspoonful every 3 hours.

℞ Silver nitrate solution, (10%)
S.: Apply locally to fauces.

For extreme cough and restlessness:

℞ Heroin, gr. 1/50 to 1/30
S.: Give one dose only in sweetened water.
or
Codein, gr. 1/20
S.: Give in sweetened water. May repeat once after 4 hours if needed.

(NOTE: The above doses of heroin and codein are intended for a single administration, or to be repeated not more than once. It is better to give a full sized dose that will control the cough than to repeat small doses without effect.)

For the conjunctivitis:

℞ Yellow oxide of mercury, gr. 1/2
Petrolatum, dr. 1
S.: Apply to margins of the eyelids and to the nostrils.

℞ Argyrol, dr. 2
Distilled water, oz. 1
S.: Instil a few drops into the conjunctival sac and into the nostrils.

For laryngitis:

℞ Ipecac,
Antimony and potassium tartrate,
of each gr. 1/100
Make into tablet.
S.: Give 1 tablet every 4 hours until 10 are taken.

℞ Oil of pine, dr. 1
or
Oil of Eucalyptus, dr. 1
or
Compound tincture benzoin, dr. 1
Water, pt. 1
S.: Give steam inhalations under a tent.

(NOTE: Plain steam is probably just as effective.)

MUMPS

℞ Alkaline antiseptic solution
S.: Dilute one-half to two-thirds with water and use as mouth wash.

(NOTE: Cold applications act best as analgesics.)

NEPHRITIS

As adjuvant to dietetic and hygienic measures:

℞ Sodium sulphate, gr. 10
Water, dr. 1
S.: Give 1 teaspoonful every 3 or 4 hours, as depletive, until watery evacuations are established.

℞ Diuretin, gr. 1
Water, dr. 1
S.: Give 1 teaspoonful every 4 hours, as a diuretic.

(NOTE: Diuretics are to be avoided in glomerular types of nephritis.)

During convalescence:

℞ Syrup iodid of iron m. 5
Emulsion cod-liver oil (33%), dr. 1
Flavor with simple elixir.
S.: Give 1 teaspoonful 3 times a day.

PARASITES (SKIN)

For pediculosis capitis:

℞ Tincture larkspur,
S.: Saturate hair and scalp and cover with a towel for the night.

℞ Vinegar, oz. 1
Water, pt. 1
S.: Pack overnight to loosen eggs. Follow by shampoo next morning.

For pediculosis pubis, hair and eyebrows infested:

℞ Calomel ointment or powder,
S.: Apply to hair and eyebrows.

For scabies:

℞ Hyposulphite of soda, oz. 1
Water, gal. 5
S.: Bathe child 5 or 10 minutes before applying ointment.

℞ Sulphur ointment,
Petrolatum, of each, oz. 1
S.: Apply to affected parts daily.

For ringworm (scalp):

℞ Bichloride of mercury, gr. 1
Nickel iodid, gr. 5
Benzoinated lard, oz. 1/2
S.: Apply following depilation.

(NOTE: Drugs are often ineffective in this condition. The x-ray is the best therapeutic agent.)

For ringworm (skin):

℞ Phenol, gr. 5
Salicylic acid, gr. 15
Sulphur, gr. 20
Benzoinated lard, oz. 1
S.: Apply locally following green soap cleansing.

℞ Tincture iodin,
Alcohol, of each, oz. 1/2
S.: Apply locally every other day until dermatitis is set up.

For the irritation following bites of bed-bugs and fleas:

℞ Phenol, m. 30
Calamine powder, dr. 1
Calamine lotion, to make, oz. 3
S.: Shake bottle, pour out and dab on skin with cotton.

To keep fleas and mosquitoes away:

℞ Oil pennyroyal, m. 30
Oil eucalyptus, m. 60
Oil lavender, m. 60
Glycerin, dr. 2
Alcohol, to make, oz. 1
S.: Apply to wrists, neck, ankles and waist.

PERTUSSIS

(NOTE: Vaccine as a prophylactic is valuable; as a curative is of doubtful value.)

For the cough:

℞ Antipyrin, gr. 1
Spirits chloroform, m. 2
Bromide of soda, gr. 2
Caffein sodium benzoate, gr. 1/4
Syrup of raspberry,
Water, of each to make, dr. 1
S.: Give 1 teaspoonful every 3 hours.

℞ Codein, gr. 1/20
Bromide of soda, gr. 3
Simple elixir, dr. 1
S.: Give 1 teaspoonful at bedtime; may repeat dose once after 4 hours.

For vomiting:

℞ Codein, gr. 1/20
or
Morphin, gr. 1/50
S.: To be administered once or twice with caution.

PRURITUS (ANI)

℞ Phenol, m. 10
Zinc carbonate, gr. 20
Calamine lotion, to make oz. 1
S.: Shake, pour out and apply.

(NOTE: The cause should be searched out and removed.)

For pruritus vulvæ of little girls:

℞ Anesthesin, gr. 25
Olive oil, dr. 2
Benzoinated lard, oz. 1
M.: Dissolve the anesthesin in warm olive oil.
S.: Apply locally.

RICKETS

As adjuvant to hygienic and dietetic measures:

℞ Phosphorus, gr. 1/800
Emulsion cod-liver oil (33%), dr. 1
Flavor with simple elixir.
S.: Give 1 teaspoonful 3 times a day after meals.

If anemia is moderate:

℞ Phosphorus, gr. 1/800
Syrup iodid of iron, m. 10
Emulsion cod-liver oil (33%), dr. 1
Flavor with simple elixir.
S.: Give 1 teaspoonful 3 times a day after meals.

If anemia is extreme:

℞ Cacodylate of iron, (5%) sterile in ampoules.
S.: Inject 8 minims into triceps muscle for 10 days; then on alternate days for a like period.

RHUS POISONING

Extract green rhus,
S.: 5 to 10 minims intramuscularly.

℞ Thymol di-iodid, dr. 1
Phenol, m. 30

or

Solution coal tar, m. 30
Zinc carbonate, dr. 1
Calamine lotion, to make oz. 3
S.: Shake bottle, pour out and apply on cotton dabs.

(NOTE: As the active principles of rhus toxicodendron is volatile and is probably a fatty acid, the application of greases is contraindicated. The gentle application of soap and water before the use of any prescription is undoubtedly good therapeutics. This should be followed by gentle sponging with dilute alcohol).

For the dermatitis which sometimes follows:

℞ Calamine, dr. 1
Cold cream, U.S.P., to make oz. 1
S.: Apply locally.

SCARLET FEVER

For the itching:

℞ Compound solution of cresol, oz. 1
Water, gallon 10
S.: Give daily bath.

To irrigate nose and throat:

℞ Borax, gr. 10
Water, oz. 3
S.: Use as irrigating fluid.

For toxemia:

℞ Glucose, 10 per cent
Sterile water,
S.: Inject 2 to 6 ounces intravenously.

For collapse:

℞ Adrenalin solution, 1 to 1000, m. 5
Sterile normal salt solution oz. 4
S.: Inject 4 to 8 ounces of the fluid into the peritoneum.

SCURVY

℞ Orange juice, oz. 1
or
Tomato juice, oz. 1
or
Potato cream, oz. 2
S.: Give to infant every day in divided doses as prophylactic; double or treble dose as curative measure.

SMALLPOX

℞ Iodin crystals, oz. 1/4
Iodid of potash oz. 1/4
Glycerin oz. 1
Water, gallons 5
S.: Give patient daily bath.

℞ Iodin crystals, gr. 15
Potassium iodid, gr. 15
Ichthyol, dr. 2
Water, oz. 1
S.: Apply to face under mask; reapply under fresh mask daily.

STOMATITIS

For apthous type:

℞ Borax, gr. 10
Water, oz. 1
S.: Use as spray to mouth and throat.

℞ Formalin, m. 5
Water, oz. 1
S.: Apply with cotton on applicator.

℞ Tincture iodin,
Alcohol, of each, dr. 1
S.: Apply with cotton on applicator.

For ulcerative type with gingivitis:

℞ Trichloracetic acid, 50%
S.: Lightly touch ulcers and gingival margins, daily for 2 or 3 days.

(NOTE: This is a powerful drug and care must be taken that no excess acid be allowed to injure the surrounding tissue.)

℞ Peroxide of hydrogen, dr. 2
Water, oz. 1
S.: Use as a spray.

℞ Zinc chloride, gr. 1/10
Glycerin, dr. 1
Cinnamon water, oz. 1
S.: Use as a spray.

SYNOVITIS

℞ Methyl salicylate, dr. 1
Guiacol m. 5
Benzoinated lard, oz. 1
S.: Apply to painful joints under cotton; fix joint with light splint.

℞ Methyl salicylate, gr. 3
Powdered sugar, gr. 5
S.: Give 1 dose 3 times a day.

SYPHILIS

℞ Mercurial ointment mild, gr. 20
S.: Rub in skin following hot bath.

℞ Bichloride of mercury, gr. 1/500
S.: Give in sweetened water 4 times a day.

℞ Mercury with chalk, gr. 1
Powdered sugar, gr. 5
S.: Give 1 powder 4 times a day.

℞ Arsphenamin (salvarsan), 1 decigram
S.: Inject intravenously every 10 to 20 days for 10 injections.

℞ Neoarsphenamin (neosalvarsan), 1.5 decigrams
S.: Inject intravenously or intramuscularly every 10 to 20 days for 10 injections.

For nasal discharge:

℞ Grey oil, m. 30
Paraffin oil, dr. 2
S.: Put a few drops into nostrils 2 or 3 times a day.

For rhagades and ulcers about the mouth:

℞ Black wash, dr. 2
Calamine lotion, to make oz. 1
S.: Apply locally.

℞ Mercury nitrate, gr. 1/24
Compound tincture benzoin, oz. 1
S.: Apply to lesions with cotton on applicator.

For circumanal condylomata:

℞ Phenol, m. 15
Black wash,
Milk of magnesia,
Rose water, of each, oz. 1
S.: Soak compresses in solution and apply so as to keep opposing surfaces apart.

℞ Ammoniated mercury, gr. 1
Zinc stearate, oz. 1/2
S.: Apply as dusting powder.

For mucous patches:

℞ Tincture iodin, dr. 1
Alcohol, dr. 2
S.: Apply to patches with cotton on applicator.

℞ Bichloride of mercury, gr. 1
Water, oz. 1
S.: Apply to patches with cotton on applicator.

TETANY

As adjuvant to gastric lavage, dietetic and hygienic measures:

℞ Calcium chloride, gr. 10 to 20
S.: Add to each bottle feeding or give in gelatin jelly 4 times a day.

℞ Phosphorus, gr. 1/600
Emulsion cod-liver oil (33%), dr. 1
Simple elixir to flavor.
S.: Give 1 teaspoonful 3 times a day.

TETANUS

℞ Antitetanic serum, 20 to 30 c.c.
S.: Inject daily into a vein and the same amount into the spinal canal until symptoms abate.

℞ Magnesium sulphate, gr. 4
Water, m. 15
S.: Inject intraspinally.

℞ Chloral hydrate, gr. 3
Water, oz. 1
S.: Inject into rectum following cleansing enema.

℞ Morphin sulphate, gr. 1/30
S.: Inject hypodermically.

THRUSH

℞ Sulphurous acid, m. 3
Water, dr. 1
S.: Apply gently to affected areas once a day.

℞ Sodium thiosulphate, gr. 10
Water, oz. 1
S.: Apply freely with cotton on applicator to affected parts before meals or after if it does not nauseate.

℞ Borax, gr. 30
Water, oz. 1
S.: Use as spray to mouth and throat.

TONSILLITIS

℞ Solution silver nitrate, 10%
S.: Paint tonsils daily for 2 or 3 days.

℞ Iodin, gr. 25
S.: Apply locally.
Potassium iodid, gr. 25
Glycerin,
Water, of each to make oz. 1
S.: Paint tonsils daily for 2 or 3 days.

℞ Borax, gr. 30
Water, oz. 1
S.: Use as spray.

℞ Bichloride of mercury, gr. 1/1000
Tincture chloride of iron, m. 5
Glycerin,
Water, of each to make, dr. 1
S.: Give 1 teaspoonful every 2 hours for 4 or 5 doses; then every 4 hours for a like number. Do not give near meal time.

URTICARIA

℞ Coal tar solution, (N. F.) m. 20
or
Phenol, m. 50
Zinc oxide, dr. 2
Calamine lotion, oz. 1

℞ Atropin sulphate, gr. 1/800
Mercury with chalk, gr. 1/2
Taka-diastase, gr. 2
Calcium carbonate, gr. 5
M.S.: Give one such powder 3 times a day.

℞ Calomel, gr. 1/10
S.: Give 1 tablet every 20 minutes until 1 grain has been taken.

(NOTE: The offending protein should be traced and the patient immunized by increasing doses of the same protein. It should be remembered that biting insects may be responsible for urticarial attacks by introducing a heterologous protein under the skin.)

For acute or giant urticaria:

℞ Atropin sulphate, gr. 1/800
Adrenalin, (1 to 3500), ♏ 8
S.: Inject into triceps muscle.

(NOTE: The distressing urticaria that follows serum injections is often promptly amenable to this therapy.)

VOMITING

No therapy is to be undertaken until it is certain that the vomiting is not a symptom of bowel obstruction.

Adjuvant to dietetic measures:

℞ Sodium bicarbonate, oz. 1
Water, qt. 1
S.: For stomach lavage.

℞ Sodium bicarbonate, oz. 2
Water, qt. 1
S.: For bowel lavage.

℞ Sodium bicarbonate, gr. 5
Water, oz. 1
S.: To be given frequently by mouth, well iced and in small doses.

For intractable vomiting:

℞ Sterile normal salt solution (0.8%)
S.: Inject subcutaneously or intraperitoneally.

℞ Codein, gr. 1/20
or
Morphin, gr. 1/40
S.: Give hypodermically to quiet child; may repeat in 4 hours if needed.

WARTS

℞ Salicylic acid plaster
S.: Cut size of wart and apply.

℞ Trichloracetic acid, 50%
S.: Apply daily with wood applicator using great care to prevent excess acid from injuring surrounding skin.

WORMS

For tape worms:

℞ Oleoresin male fern, gr. 5
Put into capsule.
S.: Give 1 capsule every hour for 6 doses; hold child's mouth open and the tongue down with a tongue depressor and toss capsule into pharynx.

℞ Atropin sulphate, gr. 1/800
Sodium sulphate, dr. 1
Glycerin, dr. 2
Chloroform water,
Water, of each to make oz. 2
S.: Give as a cathartic following the last dose of the vermifuge.

For round worms:

℞ Santonin, gr. 1/2
Powdered sugar, gr. 15
S.: Give 3 doses at 2 hour intervals.

(NOTE: The treatment should be initiated by a mild cathartic. The remedy should be given on an empty stomach. It should be followed by a brisk purge such as sodium sulphate or milk of magnesia. An oil cathartic should not be given following male fern.)

For thread worms:

℞ Infusion of quassia,
S.: Inject 4 to 8 ounces into rectum.

℞ Sodium chloride, dr. 1
Water, oz. 8
S.: Inject into rectum.

℞ Bichloride of mercury, 1 to 10,000.
S.: Inject 8 ounces high into colon following a sodium bicarbonate enema to remove mucus.

(NOTE: Some cases are resistant to all forms of medication by rectum. For such, give santonin as for round worms. Cleanliness about the anus must be enforced and the child's fingers should be kept scrubbed and painted with collodion to prevent transportation of eggs from anus to mouth.)

For hookworm:

℞ Thymol, gr. 1/2
Chocolate syrup, dr. 1
S.: Give 1 teaspoonful every hour for 4 doses.

or

℞ Thymol, gr. 1/2
Put in capsule.
S.: Give 1 capsule every hour for 4 doses. Hold child's mouth open and the tongue down with a tongue depressor and toss capsule into pharynx.

(NOTE: Any of the vermifuges may react very severely on some children and they should be used with caution and with the patient under strictest observation.)

CHAPTER XXV

POISONING

The physician is often called upon to prescribe for children who have, or are supposed to have taken poison. Except in comparatively rare instances in which potent drugs are dispensed in the form of confections or sugar-coated pills and carelessly left within the child's reach, the poison case usually turns out to be a case of fright on the part of the parents. However, there are instances where service cannot be too prompt and as the physician cannot know which case is going to be real and which spurious, he is never justified in regarding any case lightly.

Outside the highly potent drugs, such as opium, arsenic and strychnin, the average case of poisoning in children is mild for the reason that the child does not like the taste and after the first mouthful which may burn or excoriate the mouth and lips, he refuses to take any more. These are the cases that create the great unnecessary alarm in the household and cause the doctor a hurried visit. It should be remembered, however, that young babies are not so discriminating and many severe cases of corrosive poisoning from the drinking of lye solutions have occurred. The tendency of the toddler and runabout and even of older children to put everything that they can get their hands on into their mouths, makes the danger of poisoning a real one.

The physician usually gets the call to visit a poisoned patient by telephone. As much valuable time may be lost between the time he gets the call and the time he arrives at the bedside, it is well to instruct the parents or attendants when the call is received, regarding the first aid measures that should be used. If the variety of the ingested poison is known the appropriate antidote will suggest itself, and very often the family medicine chest or that of the neighbors contains this remedy. In the usual case of poisoning from whatever cause, an emetic is not amiss. It causes an early removal of the offending drug before there has been much time for absorption. As an emetic, mustard water fills the requirement admirably. While slightly irritating to the gastric mucosa, it is not sufficiently so to do any damage particularly if

given in dilutions of one level tablespoonful to a glass of warm water. Its effect is usually prompt. Syrup of ipecac in teaspoonful doses is usually easily available and is an effective emetic. The parents should be instructed to keep the child warm until the physician arrives.

As soon as the case is seen, stimulation should be given if it be necessary, and gastric lavage should be immediately instituted in all instances of poisoning by mouth except those produced by the corrosive acids and alkalies; and even in this latter category, the passing of a stomach tube is not contraindicated unless there is evidence of great tissue destruction. Happily during childhood, this is a very rare condition.

The statement of the parents that the child has vomited should not be considered as sufficient evidence that the stomach has been thoroughly emptied. The character of the fluid used for gastric lavage will depend somewhat on the history of the case, but usually normal salt solution or sodium bicarbonate solution, 2%, is desirable. The stomach is cleared of its contents, including the antidote which has been administered. There is no occasion for leaving the antidote in the stomach more than a few minutes. After the water returns clear, a little of the antidote may be gravitated into the stomach to go on into the intestine, or a cathartic may be administered in the same manner. The child should then be put to bed and treatment continued appropriate to the poison ingested.

In institutions that routinely treat cases of poisoning, it is well to have constantly prepared a saturated solution of the sulphate of iron in water; this should be kept in one bottle and in another should be prepared a 10% solution of calcined magnesia and 4% animal charcoal in water. These added together make a "universal antidote" to be administered to cases of unknown poisoning. These solutions should be put together and immediately be administered in ounce doses by stomach tube if need be.

In the following pages, the writers have not attempted to go exhaustively into the field of toxicology, but to discuss those cases of poisoning that are commonest and the method of treatment that has proved useful in their hands. It is endeavored to simplify the procedure by enumerating the steps to be followed one by one:

Equipment—

1. Stomach tube. (A large catheter with the eye end cut off and the edge rounded on sandpaper will serve.)
2. Glass connector.
3. Rubber tubing, 2 1/2 feet long.
4. Funnel or douche can.
5. Pitcher.
6. Solution (depending upon poison ingested).
7. Mouth-gag.

The Procedure—

1. Stimulate the patient if there is cardiac or respiratory failure. Strophanthin, camphor in oil, adrenalin, atropin, or strychnin are useful stimulants. While giving stimulation, have assistant.

2. Give antidote if there is one.

3. Administer emetic, mustard water or syrup ipecac.

4. While assembling equipment for gastric lavage, have some one wrap the child in a sheet, restraining the arms to the sides and lay him on his back on a table.

5. Insert mouth gag and pass the tube, with the air exhausted, into the stomach. In older children as in adults, the tube may be passed through the nares into the stomach if desired. With the tube in the stomach, turn the child on its side.

6. Pour irrigating fluid into the funnel and after a small amount has run in, lower the funnel to a point 1 or 2 feet below the level of the stomach and siphon out the stomach contents.

7. Elevate the funnel and run 3 to 6 ounces of fluid into the stomach and again lower the funnel. Repeat this process until the fluid returns clear.

8. With the stomach empty, gravitate an ounce or two of the antidote solution into the viscus and remove the tube. A cathartic may be administered in this way if desired.

9. Return the child to a warm bed and administer supportive treatment. If the child is stuporous, keep him awake; if in tonic or clonic convulsion, give sedatives or chloroform inhalations.

10. After the acute symptoms have passed, the treatment should be directed toward elimination by catharsis and diuresis.

The following is a list of the commoner poisons with their antidotes:

Poison	Antidote, chemical and physiological
Acetanilid	Alcohol, coffee, digitalis, atropine
Acids:	Magnesia, chalk, soap, tooth powder
Hydrochloric	Milk
Nitric	
Sulphuric	
Oxalic	
Phosphoric	
Acetic	
Alkalies:	Dilute vinegar, lemon juice, milk or egg white
Potash	
Lye	
Caustic soda	
Lime	
Ammonia	
Aconite	Tannic acid, tea, coffee, alcohol, strophanthus, atropin, digitalis, camphor in oil
Arsenic	Sesquioxide of iron, (freshly prepared by mixing solution of iron sulphate and solution of calcined magnesia). Opium
Atropin	Coffee, tea, caffein-sodium-benzoate Morphine Physostigmin, pilocarpine
Carbolic acid	Magnesium sulphate, alcohol
Chloral	Tea, coffee, strychnin
Digitalis	Opium, nitroglycerin
Lead salts	Magnesium sulphate, potassium iodid Opium
Mercurials	Albumen, (milk or egg white) Atropin, opium
Opium	Potassium permanganate Atropin Coffee, caffein-sodium-benzoate
Phosphorus	Old oil of turpentine Copper sulphate Avoid oils and fats
Silver nitrate	Sodium chloride Potassium iodid
Strychnin	Alkalies, tannic acid, tea, coffee Chloral, bromides Chloroform Alcohol Opium

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